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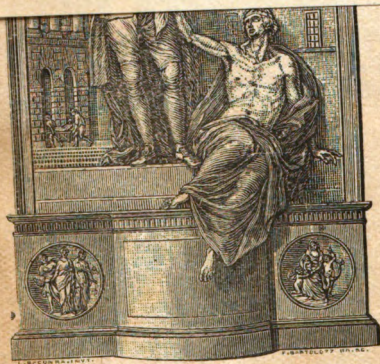
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VOL. LVI.,

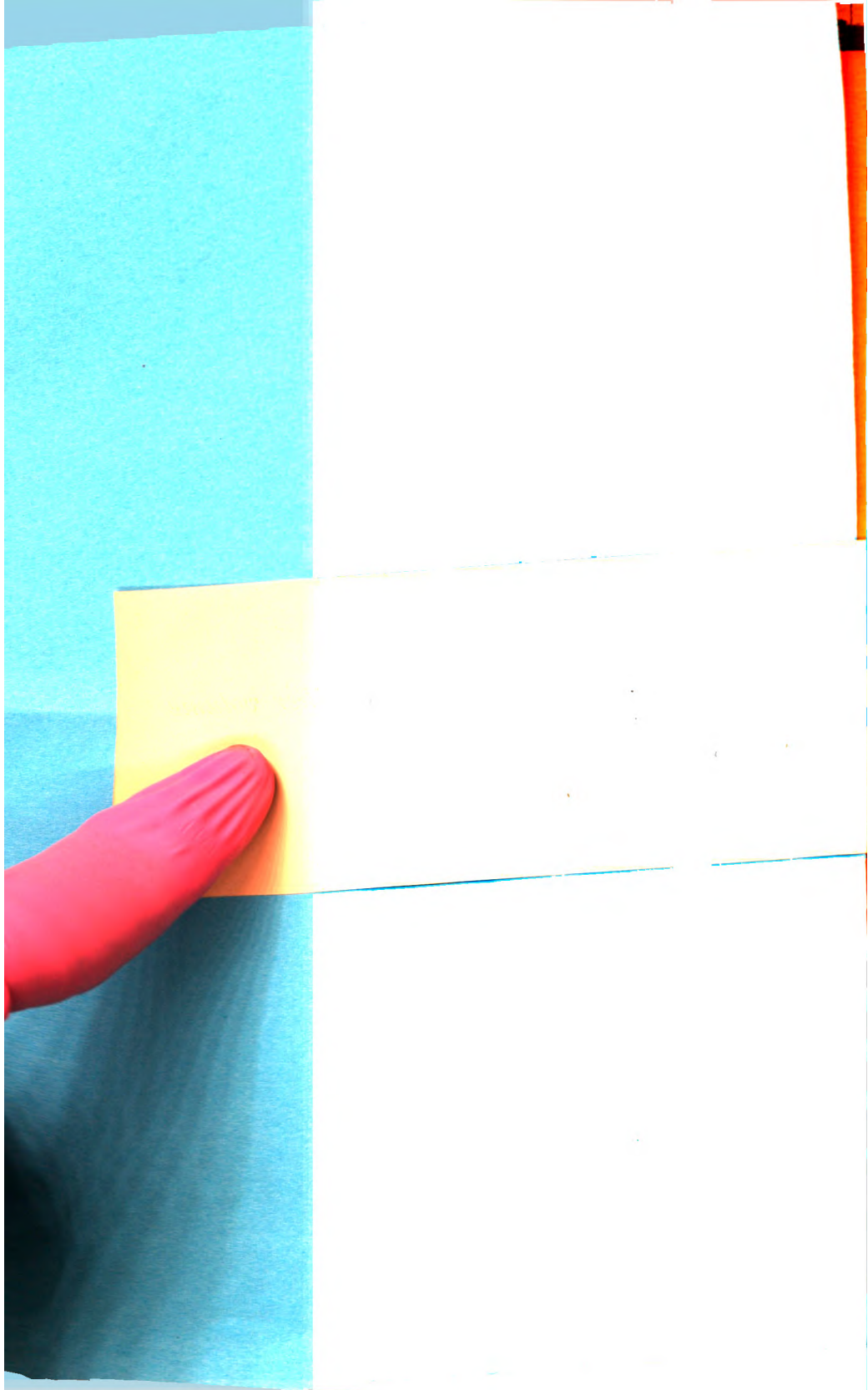
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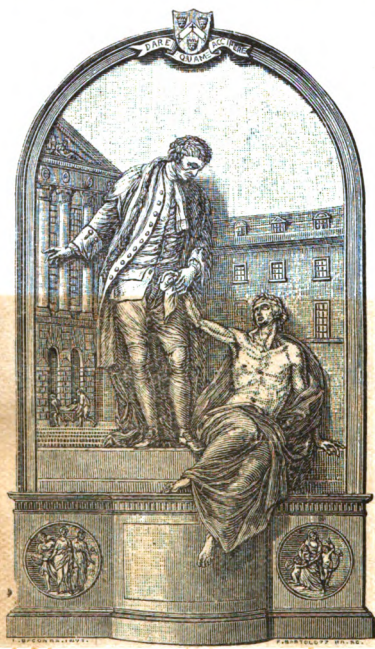
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VOL. LVI.,
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Delbruck, R. E., B.A., M.B., B.C., Embankment Chambers,
Villiers Street, Strand, W.C.
De Mierre, A., 6, Seaside Road, Eastbourne
Dencer, W. H., Guy's Hospital
Denham, N., 1, Albemarle Road, Beckenham, Kent
Denman, R., Port Victoria, Mahé, Seychelles
Denning, A. W. F., Guy's Hospital
Dennant, W. A., Guy's Hospital
Denny, Surgeon H. R. H., R.N., H.M.S. *Magnificent*, Channel
Squadron
Dent, H. L., Guy's Hospital
Denyer, C. H., Guy's Hospital
Derriman, W. E., Guy's Hospital
Desprez, H. S., Shoreham, Sevenoaks
De Villiers, C. C. A., Guy's Hospital
Dickey, W. C. McN., Guy's Hospital
Dickson, A. C., Guy's Hospital
Digby, K. H., Guy's Hospital
Dimock, E. C., 85, Victoria Road, Aldershot
Dismorr, C. J. S., Guy's Hospital
Dobson, T. H. B., M.B., Guy's Hospital
Docra, C. G., Guy's Hospital
Dodd, A. H., 49, Church Road, Hove, Brighton
Dolman, A. J., General Dispensary, Lincoln
Donald, J. A., Guy's Hospital
Donnell, J. H., Guy's Hospital
Doudney, L., Guy's Hospital
Douglas, W. T. Parker, M.B., Ivy House, Newbury, Berks
Douse, J. F., Guy's Hospital
Dowsett, E. B., 1, Gloucester Street, Portman Square, W.
Drake, G. H., 35, Old Elvet, Durham
Drew, H. W., Eastgate, East Croydon
Drewitt, A., 15, Prospect Place, Harrogate.
Du Boulay, H. H., 2, Royal Terrace, Weymouth
Du Buisson, E. W., Hereford
Duffett, H. A., Withy Holt, Sidcup
Dundee Medical Library, c/o Dr. W. E. Froggie, 30, Springfield,
Dundee
Dunn, L. A., M.S., The College, Guy's Hospital, S.E.
Duran, Carlos, and Nunez, Daniel, San José, Costa Rica
Durant, A. R., Guy's Hospital
Durham, F., M.B., 52, Brook Street, W.
Durham, H. E., M.A., M.B., B.C., Physiological Laboratory,
Cambridge

Dymott, G. L., Guy's Hospital
 Dyson, P. A. S., Guy's Hospital

Eager, Reginald, M.D., Northwoods Asylum, Frampton Cotterell,
 near Bristol

Eason, H. L., M.B., B.S., Guy's Hospital

Eastes, George, M.B., 35, Gloucester Place, Hyde Park, W.

Eastes, G. Leslie, M.B., B.Sc., 62, Queen Anne Street, S.W.

Eastes, T., M.D., 18, Manor Road, Folkestone

Eccles, H. D., Monganui, New Zealand

Edey, T. H., Guy's Hospital

Edmonds, W. H., Guy's Hospital

Edridge, Ray, Guy's Hospital

Edwards, C., City of London Asylum, near Dartford, Kent

Edwards, C. D., B.A., M.B., B.C., Alton Lodge, Woodford
 Green, Essex

Edwards, F. H., M.D., Camberwell House, Peckham Road, S.W.

Edwards, O., 34, Etnam Street, Leominster

Edwards, W. H., Trevone, Knollys Road, Streatham, S.W.

Elcum, D., M.D., 41, Montpellier Terrace, Cheltenham

Elliott, C. C., M.D., B.S., Sea Point, Cape Town, South Africa

Elliott-Blake, H., 3, Ellasdale Road, Bognor

Ellis, G. G., 49, Sandgate Road, Folkestone

Elphinstone, R., Forest House, Silverstone, Towcester, North-
 amptonshire

Elwood, W. H., Guy's Hospital

Emms, A. Wilson, J.P., M.D., Belgrave, Leicester

English, D. C., M.D., Post Office Box 87, New Brunswick, New
 Jersey, U. S. America

Ensor, C. A., Tisbury, Salisbury

Evans, Alfred H., Sutton Coldfield, Warwickshire

Evans, G., Guy's Hospital

Evans, J. H., Broomfield, Crosby Road North, Waterloo, Liver-
 pool

Evans, J. R., Ammanford, Carmarthenshire

Evans, J., Guy's Hospital

Evershed, A. R. F., 49, Knollys Road, Streatham, S.W.

Every-Clayton, L. E. V., M.D., B.S., Emsworth, Hants

Evison, F. A., March, Cambridgeshire

Ewart, J. H., Eastney, Devonshire Place, Eastbourne

Ewen, H. W., Kynance, York Avenue, East Cowes, Isle of Wight

Eyles, A. W., Guy's Hospital

Fagge, C. H., M.S., 22, St. Thomas's Street, S.E.

Fagge, R. H., High Street, Melton Mowbray

Farr, E. A., J.P., Heath House, Andover, Hants

Farrant, E., Guy's Hospital

Faulks, E., Guy's Hospital

- Fawcett, J., M.D., B.S., 26, St. Thomas's Street, S.E.
Fawley, T. B., Guy's Hospital
Fawsett, F. W., Guy's Hospital
Fawsitt, Thomas, 46, Union Street West, Oldham
Fearn, I. R., Guy's Hospital
Featherstone, J. W., Guy's Hospital
Felton, R., Guy's Hospital
Fenn, C. D., Park House, Diss, Norfolk
Field, Ernest, M.D., C.M., 8, Belmont, Bath
Field, G. H., B.A., M.B., B.C., Cedarlea, Poole Road, Bournemouth, West
Fisher, Theo., M.D., Harley Lodge, Clifton Down, Clifton, Bristol
Fisher, W. H., M.A., M.B., B.C., Oak Street, Fakenham, Norfolk
Fleury, Captain C. M., R.A.M.C., Sind Club, Karachi, Bombay, India
Floyd, S. G., M.D., B.S., Llandrindod Wells, Radnor
Forbes, A. H., Guy's Hospital
Forman, E. Baxter, M.D., 11, Bramham Gardens, South Kensington, S.W.
Forsyth, D., Guy's Hospital
Fort, H. R. T., B.A., Chorleywood, Rickmansworth
Fortescue-Brickdale, J. M., M.A., M.B., B.Ch., Uppingham, Rutland
Forty, D. H., Edbrook, Wotton-under-Edge, Gloucestershire
Foster, A. L., Guy's Hospital
Foster, C. M., M.D., 1101, Yonge Street, Toronto, Canada
Foster, O. H., M.A., M.B., Hitchin, Hertfordshire
Fountaine, D. O., c/o T. George, Esq., Pollicott, Thane, Oxon.
Fowke, F. W., Byfield, R.S.O., Northants
Fox, A. D., Guy's Hospital
Fox, F. N., Guy's Hospital
Fox, H. E. C., M.B., Surgeon R.N., H.M.S. *Duke of Wellington*, Portsmouth
Fox, H. J., Guy's Hospital
Fox, J. A., Tregea House, Penzance
Francis, J. S., Guy's Hospital
Frankenburg, L. H., B.A., Guy's Hospital
Franklin, R., Guy's Hospital
Fraser, A., M.B., 22, Shooters Hill Road, Blackheath, S.E.
Fraser, C. F., Guy's Hospital
Fraser, J. A., Western Lodge, Romford, Essex
Frazer, E. E., M.D., B.S., Helena House, Great Union Road, St. Heliers, Jersey
Fremantle, F. E., B.A., M.B., B.Ch., 39, Moore Street, Lennox Gardens, S.W.
French, H. S., B.A., M.B., B.Ch., Guy's Hospital
Fripp, A. D., C.B., C.V.O., M.S., 19, Portland Place, W.

- Frost, E. B. M., 216, Queen's Road, New Cross, S.E.
 Fry, A. Cradock, B.A., M.B., Priory House, Wellesley Road,
 Colchester
 Fry, J. F., Shepton Mallet, Somersetshire
 Fuller, Courtenay J., 33, Nightingale Place, Woolwich
 Fuller, W. A., Guy's Hospital

 Gabriel, A. M., Hazlewood Lodge, Enfield
 Gaffney, E. J., Guy's Hospital
 Galabin, A. L., M.A., M.D., 49, Wimpole Street, W.
 Galton, J. H., M.D., Chunam, 14, Sylvan Road, Norwood, S.E.
 Gannev, W. E., 4, Fairfield Road, Old Charlton, Kent
 Gardner, A. L., Guy's Hospital
 Gardiner, J. J., Guy's Hospital
 Gardiner, J. N., B.A., M.B., B.C., Glenwood, Auckland Road,
 Upper Norwood, S.E.
 Gardner, P. H., Five Ways, Torquay
 Garner, W. L., B.A., M.B., B.C., The Limes, Amptill, Beds.
 Garrard, C. R. O., 30, Broad Street, Pendleton, Salford
 Garrard, W. A., Chatham House, Rotherham, Yorkshire
 Gater, A. W., Guy's Hospital
 Gathergood, B. W., M.D., Terrington St. John's, Lynn, Norfolk
 George, A. L., Guy's Hospital
 German, H. B., Guy's Hospital
 Gibson, F. G., M.A., Guy's Hospital
 Gibson, J. H., 1, Lansdown Road, Aldershot
 Giles, W., Guy's Hospital
 Gilford, H., Norwood House, Reading
 Gill, J. McD., M.D., 18, College Street, Hyde Park, Sydney,
 New South Wales
 Gillibrand, F. J., M.A., M.B., B.C., 21, Albert Road, Southport
 Gillingham, A., 485, High Road, Chiswick
 Glanville, L. S. H., Guy's Hospital
 Glendining, B., Guy's Hospital
 Glendining, R., Guy's Hospital
 Glenn, C. H., B.A., M.B., B.C., Guy's Hospital
 Giesen, E. W., Guy's Hospital
 Glover, J. A., M.B., West Malling, Kent
 Glover, J. Alison, B.A., M.B., B.C., Guy's Hospital
 Goadby, K. W., The Mall, Wanstead, N.E.
 Goble, E. W., Guy's Hospital
 Goble, F. G., Guy's Hospital
 Godson, A. H., B.A., M.B., B.C., 63, Union Street West, Oldham
 Godson, F. A., 7, Station Road, Cheadle Hulme, near Stockport
 Godson, J. H., B.A., M.B., B.C., Bank House, Cheadle, Cheshire
 Goldie, E. G., Guy's Hospital
 Golding-Bird, C. H., M.B., 12, Queen Anne Street, Cavendish
 Square, W

- Goldstein, H. M., Guy's Hospital
Goodall, E. W., M.D., Homerton Fever Hospital, N.
Goodey, A., Guy's Hospital
Goodhart, J. F., M.D., 25, Portland Place, W.
Goodman, W. J., Guy's Hospital
Goss, J., B.A., Guy's Hospital
Gosse, H. W., Eccleshall, Staffordshire
Gowing, Benjamin C., Weirfield, Penistone, Yorkshire
Goyder, David, M.D., 88, Great Horton Road, Bradford, Yorks
Graham, G. H., M.D., Buckingham Gate Gardens, James Street,
S.W.
Graham-Smith, G. S., B.A., M.B., B.C., Guy's Hospital
Granger, E. B., Little Milton, Tetsworth, Oxon
Gray, A. C. H., Guy's Hospital
Greaves, E. H., Amersham, Bucks
Green, A., M.B., Burlington Street, Chesterfield, Derbyshire
Green, A. W., 4, Wardrobe Place, St. Paul's Churchyard, E.C.
Green, N. W., Guy's Hospital
Green, R. J., Guy's Hospital
Green, T. J., Guy's Hospital
Greene, J. A. C., Guy's Hospital
Greenfield, D. G., M.B., Guy's Hospital
Greening, G. F., Guy's Hospital
Greenwood, E. Climson, 19, St. John's Wood Park, N.W.
Greenwood, J. H., 8, Park Road, Kingston-on-Thames
Greenwood, P., 155, High Street, West Norwood, S.E.
Greeves, R. A., Guy's Hospital
Grellet, H. R., Guy's Hospital
Griffin, E. H., B.A., Guy's Hospital
Griffin, T. H., Guy's Hospital
Griffin, W. E., Guy's Hospital
Griffith, H. D., Guy's Hospital
Griffith, O. Wynne, 3, Church Place, Pwllheli, N. Wales
Gromitt, J. W., Guy's Hospital
Grose, H. N., Guy's Hospital
Grove, W. Reginald, B.A., M.B., B.C., St. Ives, Hunts
Groves, C. E., F.R.S., Kennington Green, S.E.
Growse, J. L., Bildeston, Suffolk
Growse, W., B.A., Dudley House, Kenilworth
Gruggen, W., 7, Grosvenor Road, Watford
Guy's Hospital Library (Two Copies)
Guy's Hospital Museum (c/o Curator)
Gwatkin, A. J., 49, Grand Parade, Brighton
Gwynn, S. T., M.D., St. Mary's House, Whitechurch, Salop
Gwyther, H. W., Guy's Hospital

Habershon, S. H., M.D., 88, Harley Street, Cavendish Square,
W.

- Hall, F. W., M.D., M.S., 18, College Street, Hyde Park, Sydney,
New South Wales
- Hall, Surgeon R. W. B., H.M.S. 1, Pelham Road, Southsea
- Hamilton, E. T. E., M.D., M.S., B.Sc., Post Office Box 1750,
Johannesburg, Transvaal, South Africa
- Hamilton, G., Guy's Hospital
- Hammond, J. A. B., M.B., Guy's Hospital
- Hamond, P. W., Guy's Hospital
- Hancock, W. I., 7, Royal York Villas, Clifton, Bristol.
- Handley, W. S., M.D., M.S., 41, Devonshire Street, W.
- Hardenberg, E. F. H., M.B., Duffield House, Upton Road,
Watford
- Hardy, G. F., Guy's Hospital
- Hare, Major E. C., Indian Medical Service, Gauhati, Assam,
India
- Harland, G. B., Guy's Hospital
- Harnett, C. J., M.D., Atlas House, Parade, Margate
- Harper, R. S., Guy's Hospital
- Harries, T. D., Grosvenor House, Aberystwith
- Harrington, R. G., Guy's Hospital
- Harris, E. B., 1, Holy Innocents' Road, Tottenham Lane,
Hornsey, N.
- Harris, J., M.D., B.S., 127, Elizabeth Street, Hyde Park, Sydney,
New South Wales
- Harris, R., M.B., 18, Duke Street, Southport
- Harris, W. J., B.A., M.D., B.C., 37, Bell Street, Shaftesbury
- Harrison, A. J., M.B., Failand Lodge, Guthrie Road, Clifton,
Bristol
- Harrison, E. M., Guy's Hospital
- Harrison, W. W., 115, Ditchling Road, Brighton
- Harsant, J. G., M.D., The Hive, Exeter Road, Bournemouth
- Harsant, W. H., Tower House, Pembroke Road, Clifton, Bristol
- Harvey, C. P., Guy's Hospital
- Harvey, J. S. S., M.D., 1, Astwood Road, Cromwell Road, South
Kensington, S.W.
- Harvey, T. R., Guy's Hospital
- Hawkins, H., Broxbourne, Herts
- Hayes, R. H., 4, Courtfield Road, Gloucester Road, S.W.
- Hayes, T. E. D., M.D., High Street, Reigate
- Hayward, John W., Whitstable, Kent
- Hazell, F., M.B., B.S., 1, Bouquet Street, Cape Town, South
Africa
- Heap, E. F. G. T., Guy's Hospital
- Heatherley, F., M.B., B.S., Endellion, New Ferry, Cheshire
- Heddy, William Jackson, 46, Redcliffe Gardens, S.W.
- Helm, J. K. A., Guy's Hospital
- Henderson, E. E., B.A., M.B., B.C., 12, Kensington Square, W.
- Henderson, H. J., Guy's Hospital

Henderson, W., Guy's Hospital
Henson, W. J., Elmsett Hall, Wedmore, Weston-super-Mare
Hertz, A. F., B.A., Guy's Hospital
Hetley, Henry, M.D., Beaufort House, Church Road, Norwood,
S.E.
Hewetson, Captain H., R.A.M.C., Station Hospital, Rangoon,
Burma
Hickes, C., Guy's Hospital
Hickes, P. L., Guy's Hospital
Hickman, H. V., M.B., Overton House, Wanstead, N.E.
Hicks, J. T., Guy's Hospital
Hicks, R. G., 4, Paragon, Ramsgate
Higgins, C., 52, Brook Street, W.
Hilbers, H., 49, Montpelier Road, Brighton
Hillier, H. N., 19, Waterloo Place, Leamington Spa
Hills, A. Phillips, Carlton House, Prince of Wales Road, Batter-
sea Park, S.W.
Hills, H. J., Guy's Hospital
Hills, William Charles, M.D., The Chantry, Norwich
Hilton, C. T., M.B., B.S., Guy's Hospital
Hinchliff, C. J., 211, Selhurst Road, South Norwood
Hind, Wheelton, M.D., Roxeth House, Stoke-on-Trent
Hindle, F. T., Hill Croft, Askerne, Doncaster
Hinton, J. H., Guy's Hospital
Hirsch, L., Guy's Hospital
Hitchins, F. C., St. Austell, Cornwall
Hobson, J. M., M.D., Glendalough, Morland Road, Croydon
Hodgson, C. R., M.D., B.S., Marlborough House, Balham Hill,
S.W.
Hodgson, S., 201, Brixton Hill, S.W.
Hodson, Frederick, Hornsea, Hull
Hodson, J. E., Guy's Hospital
Hogarth, B. W., M.D., B.S., 11, Erving Terrace, Morecambe
Hogarth, F. W., Guy's Hospital
Hogg, R. Bowen, Timaru, Canterbury, New Zealand
Holden, H. C., Workington Hall, Cumberland
Holford, T. C., Guy's Hospital
Hollist, G. W. C., Guy's Hospital
Holloway, S. F., Holmwood, Bedford Park, W.
Holman, A. E., Guy's Hospital
Holman, C., M.D., J.P., 26, Gloucester Place, Portman Square, W.
Holman, F. K., Galeston, Eton Avenue, South Hampstead, N.W.
Holman, H. J., 1, Hardwick Road, Eastbourne
Holmes, T., M.B., Holm Lea, Forton, near Garstang
Holmes, T. E., B.A., M.B., B.C., Guy's Hospital
Hood, Donald W. C., M.D., 43, Green Street, Park Lane, W.
Hooper, H. P., Guy's Hospital
Hope, W. B., Caversham Lodge, Gosbrook Road, Caversham,
Oxon

- Hopkins, C. L., B.A., M.B., B.C., Kent County Asylum, Barming
 Heath, Maidstone
 Hopkins, F. G., M.A., M.B., B.Sc., Lynfield, Tenison Avenue,
 Cambridge
 Hopson, M. F., Grove House, Rosslyn Hill, Hampstead
 Horrocks, P., M.D., 45, Brook Street, W.
 Horsley, C. D., B.A., Guy's Hospital
 Horsley, H., 60, London Road, Croydon
 Horton, J. H., c/o W. Watson & Co., Bombay
 Houghton, N. N. A., Guy's Hospital
 Houchin, V. S., Guy's Hospital
 Howard, C. R., B.A., Guy's Hospital
 Howard, J. A., M.D., 8, Herne Hill, S.E.
 Howard, R., M.A., M.B., B.Ch., Devon House, Buckhurst Hill,
 Essex
 Howard, Wilfred, New Buckenham, Norfolk
 Howe, J. D., Deepdale House, Burrow Road, Preston
 Howell, J., M.B., B.S., General Hospital, Cheltenham
 Howell, J. B., 86, North Side, Wandsworth Common, S.W.
 Howell, T. A. I., Cleeve House, West Hill, Wandsworth, S.W.
 Howse, H. G., M.S., 59, Brook Street, W.
 Hubbard, Thos. Wells, Barming Place, Maidstone
 Hudson, A. B., Vine House, Cobham, Surrey
 Hughes, F. P., Guy's Hospital
 Hugill, George F., 32, Bedford Hill, Balham, S.W.
 Hull Medical Society, c/o Dr. J. McNidder, Anlaby Road, Hull
 Humphreys, F. R., 27, Fellows Road, N.W.
 Humphreys, G. F., Guy's Hospital
 Huntley, E., M.B., B.S., 8, Trinity Road, Tulse Hill, S.E.
 Hutchinson, F. E., Belgrave, Leicester
 Hyslop, T. B., M.D., C.M., Bethlem Royal Hospital, S.E.

 Ince, Lieut.-Colonel John, M.D., Montague House, Swanley, Kent
 Ingram, P. C. P., Guy's Hospital
 Iredell, A. W., Guy's Hospital
 Iredell, C. E., Guy's Hospital
 Isaacs, D., B.A., Guy's Hospital

 Jackson, F. D. S., Guy's Hospital
 Jackson, P. J., 216, Great Dover Street, S.E.
 Jackson, T. L., B.A., M.B., B.C., Cheadle, Cheshire
 Jackson, T. S., Fleatham, Liss, Hants
 Jacobs, J. M. C., 21, Castle Street, Shrewsbury
 Jacobson, T. B., Sleaford, Lincolnshire
 Jacobson, W. H. A., M.A., M.Ch., 66, Great Cumberland Place,
 Hyde Park, W.
 Jalland, W. H., D.L., J.P., St. Leonard's House, York
 James, Newton, Guy's Hospital

- Jaynes, V. A., 157, Jamaica Road, S.E.
 Jenkins, H. H., Guy's Hospital
 Jephcott, C., M.A., M.B., B.C., 8, The Northgate, Chester
 Jiménez, R., Guy's Hospital
 Jiménez, J. J., San José, Costa Rica, Central America
 Johnson, H., Guy's Hospital
 Johnson, R. H. C., Guy's Hospital
 Jones, A. G., Guy's Hospital
 Jones, B., M.D., Leigh, Lancashire
 Jones, F. Felix, Llanfyllin, near Oswestry
 Jones, G. H. West, Southgate, Eckington, Derbyshire
 Jones, H. J., 167, Lillie Road, S.W.
 Jones, H. S., Guy's Hospital
 Jones, H. W., Guy's Hospital
 Jones, J. Edwards, D.L., J.P., M.D., Bryn-y-ffynon, Dolgelly,
 North Wales
 Jones, Robert, 11, Nelson Street, Liverpool
 Jones, R. W., Guy's Hospital
 Jones, Surgeon Murray P., R.N., H.M.S. *Wildfire*, Sheerness
 Jones, S. H., 9, Wimpole Street, W.
 Jones, W. Makeig, M.D., Beaumont, Torquay
 Jones, W. W. C., Guy's Hospital
 Joslen, H., "Ducie," Chapelton, P.O., Jamaica
 Joy, G. P., Guy's Hospital
 Judson, T. R., 44, Mill Lane, West Derby, Liverpool
 Jupp, E. N., Guy's Hospital

 Reall, C. A. H., 31, Clarendon Road, Holland Park, W.
 Keates, H. C., M.B., Guy's Hospital
 Keele, S., 8, Highbury Place, N.
 Kellock, W. B., M.D., 94, Stamford Hill, N.
 Kelsey, Surgeon A. E., R.N., Redhill, Surrey
 Kemp, G. L., M.D., Worksop, Notts
 Kempe, A. M., 53, Church Road, Bournemouth
 Kendall, G., Battle, Sussex
 Kendall, Walter B., Greenheys, King's Wear, Devon
 Ker, Hugh Richard, Tintern, 2, Balham Hill, S.W.
 Ker, W. P., Guy's Hospital
 Kerby, R. J., Speenhamland, Newbury
 Key, B. W. M. A., B.A., M.B., B.C., 67, Victoria Road North,
 Southsea
 Kidd, W. A., M.D., B.S., 12, Montpelier Row, Blackheath, S.E.
 King, T. W., M.D., Purbrook, Dorking
 Kingcombe, A. P., Towcester, Northamptonshire
 Kingsford, E. C., Carntyne, Brondesbury, N.W.
 Kinsey-Morgan, A., M.D., 1, Stanhope Gardens, Bournemouth
 Kitchin, E. H., Guy's Hospital
 Kitching, C. M., M.D., 10, New Street, Cape Town, South Africa

- Kliszczewski, C. S., Guy's Hospital
 Knaggs, R. Lawford, M.A., M.D., M.C., 27, Park Square, Leeds
 Knight, F. C. R. M., Guy's Hospital
 Knight, H. S., Guy's Hospital
 Knowles, C. H., Casamajor Road, Egmore, Madras
 Knowles, G. F., Guy's Hospital
 Kynaston, A. E. F., Guy's Hospital

 Lacey, B. W., Guy's Hospital
 Lacey, E. E., Guy's Hospital
 Lacey, T. Warner, M.D., 26, Nightingale Place, Woolwich
 Lacey, W. J. M., Clyde House, 6, Ware Road, Hertford
 Lamb, C. J., Guy's Hospital
 Lamb, W. H., M.B., 23, Palace Court, Bayswater Hill, W.
 Lambert, A. L., 98, Montpelier Road, Brighton
 Lancaster, H. F., M.D., 154, Westbourne Terrace W.
 Lancereaux, E., M.D., 44, Rue de la Bienfaisance, Paris
 Lancet, The, 423, Strand, W.C.
 Landon, E. E. B., Bradbourn House, Acton, W.
 Landon, T. W. H., Hackbridge, Carshalton
 Lane, W. Arbuthnot, M.S., 21, Cavendish Square, W.
 Langdale, H. M., Guy's Hospital
 Lansdale, W., M.D., 44, Trinity Square, S.E.
 Lansdown, R. G. P., M.D., B.S., 39, Oakfield Road, Clifton, Bristol
 Larkin, F. G., Grove Park, Lee, Kent
 Larkin, R., Guy's Hospital
 Larking, A. E., M.D., 4, London Street, Folkestone
 Latimer, H. A., M.D., 202, St. Helens Road, Swansea
 Lavers, N., M.D., Camberwell House, Peckham Road, S.E.
 Lawry, R. C., Guy's Hospital
 Layton, T. B., Guy's Hospital
 Leader, H., M.B., 407, Fulwood Road, Ranmoor, Sheffield
 Leathes, J. B., B.A., M.B., B.Ch., St. Thomas's Hospital, S.E.
 Leckie, M., Guy's Hospital
 Ledger, A. V., Guy's Hospital
 Leeds School of Medicine Library (per the Secretary of Yorkshire
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 Leeming, A., Guy's Hospital
 Leigh, W. W., J.P., Treharris, R.S.O., South Wales
 Lewin, G., Guy's Hospital
 Lewis, A. C., Wingham, near Dover
 Lewis, R. P., Guy's Hospital
 Lewis, W. C., Guy's Hospital
 Lidderdale, F. J., M.B., B.C., Grove Hospital, Tooting, S.W.
 Lipscomb, E. H., M.B., St. Alban's, Herts
 Lipscomb, E. R. S., Starcross, Exeter
 Lister, T. D., M.D., B.S., 95, Wimpole Street
 Litchfield, P. C., Guy's Hospital

- Littlejohns, A. S., Guy's Hospital
Littlewood, J. O., West Hill Drive, Mansfield, Nottinghamshire
Lloyd, E., B.A., Guy's Hospital
Lloyd, M., M.D., Vale Villa, Llanarthney, R.S.O., Wales
Lockwood, J. P., Faringdon, Berkshire
Lockyer, G. E., Purley Rectory, Maldon, Essex
Long, D. S., B.A., M.D., B.C., 71, Micklegate, York
Longhurst, E. A., Guy's Hospital
Longson, F. M., Guy's Hospital
Loosely, W. H., 14, Stratford Place, W.
Loud, F., Albion House, Lewes
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Swayne, W. C., M.D., 8, Leicester Place, St. Paul's Road, Clifton, Bristol
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ON A METHOD OF STRETCHING, DIVIDING, OR EXCISING A PORTION OF THE LINGUAL NERVE, WITH CASES.

By R. CLEMENT LUCAS, B.S.

SURGEON TO GUY'S HOSPITAL.

In the *British Medical Journal* of November 15th, 1884, there appeared an editorial note¹ on a method of stretching the lingual nerve, which I devised for a case of intense neuralgia of the tongue, after failing to give permanent relief by division of the nerve, when following the older method suggested by Mr. Hilton for the relief of pain due to cancer of the tongue.

The progress made in the methods of removing the tongue for cancer has practically eliminated the cases for which Mr. Hilton originally suggested division of the lingual nerve.² In those days of conservative surgery, when the lingual artery—trivial little vessel that it is—was held up as a terror to surgeons, and when the operation of placing a ligature upon it was deemed of tragic importance, when surgeons feared to approach the tongue with knife or scissors and were satisfied that caustic or cautery were the only available remedies—when the presence of a cancerous gland was regarded as a sign that no operation could be of avail—in those primitive days of elementary

¹ Brit. Med. Journ., Vol. ii., 1884, p. 975.

² Guy's Hospital Reports, 1850, p. 253.

surgery, the idea of Mr. Hilton that temporary relief from the agonizing pain of tongue-cancer might be brought about by division of the lingual nerve, came and lived as a valuable suggestion, but does not appear to have been acted upon by others till ten or twelve years later. I take it for granted that now there lives not a surgeon who with a case of cancer of the tongue before him would dream of performing a palliative operation. His thoughts would be concentrated on removing as thoroughly as possible the growing disease, and even in the most advanced cases, where vitality was not actually on the ebb, he would proceed by excision to relieve the patient of the reeking decomposing mass which prevents him from taking nourishment, and by its fœtor renders him objectionable both to himself and to his friends.

Although the cases for which Mr. Hilton suggested neurotomy of the lingual have passed into another category, there still remain certain other conditions which call urgently for surgical interference. They are happily rare, but anyone who has witnessed the intense agony and distress caused by a chronic neuralgic condition of the tongue will know how anxious such patients are to be relieved of the maddening pain. The etiology of such cases is often obscure. Some, no doubt, may be found associated with a gouty diathesis, others with syphilis, but in many cases the medical attendant will be entirely at a loss to explain the cause of the intense recurring agony. Every possible remedy will have been tried without avail. Anti-podagric and anti-syphilitic remedies will have been poured in, every nerve tonic and every anodyne tried, all the soporifics administered in succession, but the patient only wakes to renewed distress. Solid food cannot be taken into the mouth without exciting stabbing pains and salivation, and the patient gradually wastes from lack of sleep and nourishment. What other causes than those mentioned may be at work to produce such painful results must generally be a matter of conjecture. Neuritis following typhoid fever or influenza is a condition which apparently picks out special nerves indiscriminately, and some of these cases may have such origin. Outgrowths of bone about the foramen ovale

may be another possible cause, but I am inclined to think that the throbbing lancinating pain so often experienced in these cases must be dependent on the throb of the companion artery on the sensitive nerve. So long ago as in my student days, in a prize essay before the Guy's Physical Society, I drew attention to the interesting fact that almost every nerve of the head and face subject to neuralgic pain passed through a small bony aperture or canal in company with a small artery. Differences in the calibre of this small artery, I argued, might be as much the cause of neuralgic pain by pressing the nerve against the sides of the bony foramen or canal, as alterations of function or pathological conditions affecting the nerve itself. In the young, functional sympathetic disturbances frequently disturb the calibre of the vessels; and in the aged, degenerative changes in the wall of the vessel might lead to similar consequences. How far these various suggestions may be justified, apart from clinical observation, it is difficult to forecast, for proper pathological investigation of such cases is most difficult ever to obtain. This is certain, whatever may be the cause, nothing short of surgical operation will in the majority of cases bring about any permanent relief.

It was for one of these cases of agonising neuralgia that I first operated by dividing the lingual nerve after the manner described by Mr. Hilton, a procedure which I found both difficult to perform and unsatisfactory in its results. But subsequently, when the nerve had united, I devised and carried out the operation it is the object of this paper to bring into prominence, which is extremely simple to perform and highly beneficial in its effect.

HILTON'S OPERATION.

Mr. Hilton thus describes the operation that he performed: "My dresser, Mr. Morgan, drew the tongue (which was much congested with blood) forwards, upwards, and towards the patient's right side. I then divided vertically, with a small knife, the mucous membrane, and submucous tissue to about three quarters of an inch in length, opposite the molar teeth; over the hyo-glossus muscle, and across the position of the sublingual gland; the progress of the operation was much impeded by the almost constant flow of blood, chiefly venous, into the wound;

but by continuing deeply by the side of the tongue this first incision, through the upper edge of the sublingual gland, I exposed the nerve, and laying hold of it with forceps I divided it with scissors. . . . The bleeding was stopped by a piece of sponge dipped in a strong solution of alum, being forced into the wound. Exactly one month from the date of the division of the nerve she began to feel again on the left side at the tip of the tongue."

It will be noticed that this operation, allowing for the increased difficulty caused by the presence of a cancerous growth, proved to be one not easy of performance, that a vertical incision three-quarters of an inch in length was made across the course of the nerve, and that it had to be carried deeply by the side of the tongue and through the upper edge of the sub-lingual gland before the nerve was reached. The operation was complicated by free hæmorrhage, which was only stopped by plugging the wound with a sponge steeped in a strong solution of alum.

MOORE'S OPERATION.

Twelve years after Mr. Hilton's suggestion was made Mr. Charles H. Moore, of the Middlesex Hospital, again drew attention to the temporary relief from pain that might be obtained by dividing the lingual nerve or nerves in cases of cancer of the tongue.³

He, however, recommended an improved method of dividing the nerve at a point further back than that at which Mr. Hilton operated. He describes his operation as follows: "The guide to the nerve in the latter situation is the last molar tooth. On passing the finger into the mouth within and beyond that, the bulging alveolar ridge can be felt narrowing as it ascends into the thin coronoid process. Behind, below, and parallel with the ridge, is the nerve. A line drawn inside the lower jaw, from the crown of the last molar tooth to the angle of the jaw, would cross it at right angles about half an inch from the tooth. An incision, therefore, in the direction of such a line, three-fourths of an inch in length, and carried through the mucous membrane to the inner surface of the bone, must divide the nerve. It is advisable to operate with a curved bistoury, since the alveolar ridge would

³ Med. Chir. Trans., vol. xlv., 1862, p. 47.

shield the nerve from the edge of a straight knife. It is also advisable to be careful in observing the position of the alveolar ridge, or, in its absence the edge of the toothless gum curving up to the ramus. In one of my earlier cases, in which there were no teeth to serve as a guide, I cut too far back and missed the nerve, being probably misled by a mass of the tumour which was adherent to the inner side of the lower jaw."

This operation would appear to be a decided advance on that originally carried out by Mr. Hilton, inasmuch as a definite guide is pointed out for finding the nerve, and the trunk of the nerve is divided farther back. But it presents several disadvantages which render its successful performance doubtful. In the first place, the "bulging alveolar ridge" caused by the last molar tooth, is only present when the tooth remains *in situ*, and in Mr. Moore's own experience, when "there were no teeth to serve as a guide," he himself failed in his endeavour to divide the nerve. With the extraction or loss of the last molar, and consequent absorption of the alveolus, Moore's guide is lost and a blind cut is made over the supposed position of the nerve. Another defect in Moore's operation is that he does not expose the nerve to view, so that whether or not the operation has been satisfactorily completed cannot be ascertained until after the patient has recovered from the anæsthetic.

THE AUTHOR'S OPERATION.

The operation which I devised seventeen years ago, but the exact details of which have never been published, depends for its accurate performance upon the anatomical fact that the lingual nerve passes from the outside of the internal pterygoid muscle to the lateral aspect of the tongue. When the jaws are widely separated the anterior edge of the internal pterygoid muscle is put on the stretch and holds the nerve outwards in contact with the jaw. If now the tongue be forcibly drawn out of the mouth and towards the opposite ear, the edge of the pterygoid muscle forms a *point d'appui* from which the nerve can be made tense. Under these conditions the nerve forms a tense bow-string under the mucous membrane, which can be both seen and felt. Thrown thus into prominence its position becomes so evident that it

cannot be missed, and it is completely under the control of the surgeon.

The details of the operation are as follows:—A sand pillow is placed under the hollow of the patient's neck, so that the mouth can be freely opened. A Gowan's or Mason's gag is introduced on the opposite side to that on which the operation is to be performed, to fix the jaw open, and a blunt hook retractor, whose prongs are an inch and a half apart, is used to widen the mouth and retract the cheek on the side of the operation. Next a stout ligature is run through the tip of the tongue close to the septum, but on the side of the operation, so that no pain may be felt in this wound after the nerve has been stretched. This ligature is tied in a loop and is used to draw the tongue forcibly out of the mouth towards the ear of the opposite side. A tense fold of mucous membrane then appears running from the inner aspect of the jaw along the side of the tongue, with a hollow above and below. This fold is caused by the stretched nerve which lies in the free edge of the fold. Should swelling prevent the appearance of the fold when the tongue is forcibly drawn to the other side, the tense nerve can still be readily felt beneath the mucous membrane with the forefinger.

The next stage is to pass a fine sharp hook, such as is used in cleft palate operations, from below upwards through the fold of mucous membrane and under the nerve. In this way the nerve is securely held. An incision about a quarter or half an inch in length through the mucous membrane, in a line with the nerve, then exposes the trunk; a blunt aneurysm needle is passed under the nerve and the sharp hook removed. The nerve is now completely isolated and may be divided, excised, or stretched according to the surgeon's views as to what will best relieve the patient.

The steps of the operation, as just detailed, were first worked out on the dead subject and implicitly followed in the first operation performed in 1884. Subsequently, I have not generally troubled to use the sharp hook, as if the tongue be sufficiently tense, the nerve cannot be missed in cutting through the mucous

membrane over it. A fine suture may be used to close the small wound in the mucous membrane.

Performed in this way the operation is practically bloodless, and compares very favourably with the incision across the course of the nerve into the side of the tongue and upper part of the sub-lingual gland, as performed by Mr. Hilton.

It is impossible, if the details are followed out, that the surgeon can fail, for the operation rests on an unalterable anatomical fact, the relation of the nerve to the internal pterygoid muscle and the side of the tongue. In this respect it is very superior to Moore's operation, which depends on a guide which is not constant, the alveolar prominence below the last molar tooth disappearing with the loss of that tooth. Moreover, as I before pointed out, Moore's operation is a blind one, for no attempt is made to expose the nerve, and it can only be a conjecture, till after recovery from the anæsthetic, that the nerve has actually been divided. Further, it is inapplicable to stretching of the nerve, which I maintain is a far more satisfactory operation than division, being more lasting in its effects, and allowing of repetition should the pain recur.

An account of this operation was given by the late Mr. W. Morrant Baker,⁴ in Heath's Dictionary of Practical Surgery, but I believe it did not find its way into the ordinary text-books or works on operative surgery, either then or since. Much indeed that is original or of value is apt to be lost in a dictionary, owing to the disconnected nature of adjacent articles.

The following cases illustrate the advantages of the operation :

CASE 1.—W. A., æt. 48, was admitted into Guy's Hospital, under Mr. Clement Lucas's care, on October 30th, 1884. He had suffered from both syphilis and gonorrhœa in former years, and his wife had had three miscarriages and no living child. He had suffered from tumours about his body, which came and disappeared. There were two in front of the right leg, one on the forehead, and one on the outer side of the right eye, which has been breaking down for some time. He had knocked his left leg,

⁴ A Dictionary of Practical Surgery. Edited by C. Heath. 1886, vol. i., p. 654.

which had been ulcerating for some months, in consequence. He was, however, admitted on account of an exceedingly painful condition of the right side of his tongue. He was in a medical ward a year ago on account of the painful tongue, and Mr. Lucas then saw him and divided the nerve. This gave him temporary relief, but the pain returned a few weeks later. Six or seven years ago the patient's tongue first began to trouble him, causing him great pain when eating or talking. He has suffered more or less ever since. Patient is rather pale but fairly well nourished. There is a swelling on the outer side of his right eye, an inch and a half by one inch, discharging serous fluid on the inner side. Another swelling is situated on the front of his right leg, an inch and a half in length by three-quarters of an inch, larger at some times than at others. On the outer side of this is the scar of an old swelling over the fibula, about an inch in diameter. There is also a raised lump at the junction of the middle and lower thirds of the tibia. On the front of the left tibia is a granulating sore as big as a walnut. His tongue is very red, and tender underneath on the right side. Near the base is a small tender lump about the size of a pea, which is excessively tender, and is probably the site of the former operation. The pain is much greater at one time than another. Talking and eating bring it on, and the pain seems to paralyse the tongue so that he cannot move it. He was ordered fifteen grains of iodide of potassium, with tartrate of iron three times a day.

The operation.—On November 11th, 1884, chloroform was administered, Gowan's gag was introduced between the molar teeth on the left side, and the right cheek was drawn back by a large pair of hook retractors. A stout silk ligature was next placed through the tip of the tongue, and by means of this the tongue was drawn forcibly out of the mouth and towards the left side. As a consequence the lingual nerve was made to stand out as a band beneath the mucous membrane at the posterior and lower part of the tongue, where it could be readily felt to roll under the finger. A sharp hook was next passed through the mucous membrane from below upwards underneath the nerve so as to fix it. Being held firmly by this hook, Mr. Lucas cut

through the mucous membrane over the nerve for about half an inch, then passed a blunt hook under it. The sharp hook being withdrawn, Mr. Lucas pulled forcibly on the nerve, first forwards then backwards, so as to thoroughly stretch it. There was scarcely any bleeding.

November 12th. The patient suffered scarcely any ill effect from the anæsthetic, and he has felt none of the old pain in his tongue since the operation. He can now move his tongue about without exciting any pain.

November 19th. The small operation wound has healed. He has had no pain in the tongue since the operation, and can now eat and talk with comfort. The sore on his left leg has healed and the discharge from the place near his right eye has ceased. The lump on his right shin has also gone down. He was discharged to day.

RE-ADMISSION THREE YEARS LATER.—W. A., æt. 51, was re-admitted into Job ward on June 15th, 1887, on account of a recurrence of the pain in his tongue. He was a pale, thin, worn looking man. He described the pain as if his tongue was being sawn across, and sometimes he had difficulty in swallowing. The same operation was repeated, except that Mr. Lucas did not trouble to put the sharp hook under the nerve before cutting down on it. The nerve was thoroughly stretched and he was immediately relieved of all pain.

He was discharged on June 24th, quite free from pain. He attended among my out-patients and took iodide of potassium for many months after his discharge, but no recurrence of pain was noted, and I believe the second operation resulted in a complete cure.

CASE 2.—Miss P., a maiden lady, æt. 65, was brought to see me on December 17th, 1885, by a member of the hospital staff. She was a tall, thin, delicate looking old lady, living in easy circumstances with her sister. For more than twelve months she had suffered from an acute stabbing, neuralgic pain on the left side of her tongue. It comes on whenever she talks or eats, and for some time she has been unable to swallow anything but fluid diet and sops. The pain is so intense that it causes tears

to flow from her eyes. When the pain first came on she had a badly fitting set of false teeth, but the replacing of these by others fitting correctly did not relieve the pain. At one time some pain was felt in the lips on the left side, due probably to the sympathetic irritation of the buccal branch of the third division of the fifth nerve, but this pain did not continue.

December 19th, 1885, Mr. H. Davis gave an anæsthetic and I operated in a similar way to that already described. When the nerve was exposed, and I was stretching it with an aneurysm needle, I noticed something immediately beneath it, which proved to be the duct of the submaxillary gland. The two cords could be readily traced by traction, the one to the papilla by the side of the frænum and the other to the side of the tongue. A fine catgut suture was used to close the small wound in the mucous membrane after stretching the nerve. The effect of the operation was to completely relieve her of the pain. I saw her some four years later, when she still remained free from pain, except for an occasional slight reminder. She then began to show symptoms of cancer of the bowel, of which she died some time after.

CASE 3.—F. L., a retired officer of the Indian Civil Service, consulted me May 6th, 1890, on account of intense neuralgia of the right side of his tongue. He was fifty-six years of age, plethoric and bronzed by the Indian climate. He had had syphilis many years ago, and also suffered from occasional attacks of gout. He had been a heavy smoker, but had lately given it up as it seemed to aggravate the pain in his tongue. The latter was white with chronic superficial glossitis, commonly known now as leucoplakia. There were several irregular sharp edged fangs and broken teeth, for which I sent him to a dentist to have them filed down. This palliative treatment gave him no relief, though the appearance of his tongue slightly improved. On June 2nd, 1890, I operated on his tongue, pulling it out by means of a silk suture put through the tip, cutting on to the prominent band of the lingual nerve and stretching it with an aneurysm needle passed beneath it. When he recovered from the anæsthetic he said the pain was entirely relieved. I saw this gentleman from time to time for several years and he remained

free from the pain in his tongue. He died three years ago in a fit of apoplexy.

CASE 4.—The widow of a clergyman, who had suffered a good deal of privation and much trouble, consulted me on February 16th, 1897. She was fifty-eight years of age, and since her menopause had been generally in weak health. She was a thin, pale, nervous-looking woman, who had been put to great sorrow by the extravagance of her son, and had denied herself many of the ordinary necessities of life. For the last eighteen months she had lived entirely on slop food, owing to agonising pain on the right half of her tongue. When she moved it in eating or speaking, a plunging, stabbing pain shot along the side to the tip and seemed completely to paralyse it. She had been in the habit of taking opium and chloral to gain sleep at night, and her nervous system generally was in an unsatisfactory state.

On February 22nd, I stretched her right lingual nerve by the method described, and it gave her immediate relief. It was some time before she could be persuaded to take a rational amount of food even when not suffering from pain, as she seemed to fear that mastication would cause it to return. She greatly improved in general health after the operation, and has not, I understand, had return of pain in the tongue; but, from what I hear, she has not been cured of the habit of taking opiates which was fostered by the pain she used to suffer and by the trouble she had had to endure.

The foregoing cases sufficiently illustrate the great relief that can be brought about by the operation, and the ease with which it can be performed.

CASE OF CONGENITAL CAVERNOUS
ANGEIOMA OF RIGHT HAND.
LIGATURE OF BRACHIAL, AND FIVE
MONTHS LATER, OF RADIAL AND
ULNAR ARTERIES. CURE PERMANENT
TEN YEARS LATER.

By W. H. A. JACOBSON, M.Ch.

N. A., æt. 18, was sent to me at the Royal Hospital for Children and Women, in 1891, with the following congenital condition of the right hand. The patient could give no account of the disease in its earlier stage. Her mother is dead. These cases of angiomas of the hand, or elsewhere, appear to form two groups. In one the disease is congenital and begins as a nævus. In the other the mischief first shows itself much later in life, and dates, in some cases, to an injury. But they are still cavernous angiomas, not arterio-venous aneurysms or aneurysms by anastomosis. All the fingers showed, back and front, and along their lateral margins, bluish-purple swellings, involving the skin and subcutaneous tissues. Here and there these swellings contained knotty nodules, as if phleboliths were present. The thumb was

14 *Case of Congenital Cavernous Angioma of Right Hand.*

not involved. The whole of the little and ring fingers, and the upper half of the second finger were the parts chiefly affected. The nail-bed of the index finger showed a chronic onychia, and the nail was unusually dry, discoloured, and inclined to break up longitudinally. Some of the swellings felt just like sacs full of blood, others, especially those in the palm, felt spongy, as if composed of cavernous tissue. All could be affected by pressure, being imperfectly emptied and at once filling up again. Owing to the presence of these swellings on the fingers it was impossible to say if the digital arteries were enlarged. In places, *e.g.*, on the back of the index finger, large varicose veins the size of an adult internal saphena ran upwards to spongy masses situated on the dorsum of the metacarpus, *e.g.*, over the second and third spaces. These swellings appeared to be continuous with other puffy swellings in the palm, less defined owing to the thicker coverings. When those on the dorsum were compressed, those in the palm increased in size. Elsewhere, *e.g.*, over the palmar aspect of the pisiform bone, swellings distinctly puffy but not discoloured were present. Apart from these angiomas the hand was not increased in size, measurements of the bones showing no increase over those on the left side; nor was there any change in the size and appearance of the forearm and arm.

The pulsation of the right radial and ulnar arteries was more distinct than on the opposite side. Pulsation was also visible over some of the largest of the above described spongy swellings, especially in that over the palmar aspect of the first phalanx of the middle finger. No thrill could be detected anywhere. The chief complaint of the patient was that the hand felt big and clumsy, and, when warm, intensely itchy. The puffiness interfered with her writing or using a needle; points of much importance as she was being trained as a schoolmistress. The swellings were always larger in cold weather, and when the hand was allowed to hang down. On two occasions when one of the fingers was accidentally cut, the bleeding is described as having been furious and difficult to arrest.

March 13th. Ether was given. As puncture with the fine point of the Pacquelin's cautery caused no clotting, merely free jets of

venous blood, and as the pulse in the right radial and ulnar arteries was markedly increased compared with that in the fellow vessels, the right brachial¹ was tied in the middle of the arm with a carbolised silk ligature.

The only difficulty met with in finding the artery was the large size of the basilic vein and the venæ comites. All pulsation below at once stopped. The hand became cold and the level of the masses of vascular tissue at once sank down, and to the feel became markedly flaccid. Two days later very faint pulsation could be detected in the radial artery.

March 23rd. Numerous setons of aseptic chromic gut were passed into the chief vascular masses, now flaccid, and knotted *in situ*. An incision was made over the varicose veins on the back of the hand, and when these were exposed they were found to be continuous, as was expected, by means of nævoid tissue passing through the second and third spaces with the spongy tissue in the palm. The veins were tied and removed. As much of the cavernous tissue as seemed safe was cut away, while two deep setons were passed into the spongy tissue which ran through the spaces, knotted *in situ* and brought out between the sutures which united the wound on the back of the hand.

The next few weeks, as the setons were withdrawn, fresh ones were introduced, the fingers were kept bandaged with narrow rolls of salalembroth gauze, wrung out of carbolic acid lotion (1 in 40). The patient was kept under observation for some months after the wounds had healed, in order that pressure could be maintained by means of narrow roller bandages.

In August she was readmitted. The puffy swellings and masses of spongy tissue had everywhere disappeared save on the back of the index, ring, and little fingers. A little was also present in the pulp of the ungual phalanges of the three outer fingers, some swelling, still feeling like cavernous tissue, persisting in the palm. But at each of these spots the amount of the disease

¹ It will be noticed below that in Sir W. Lawrence's case ligature of the radial and ulnar arteries failed, though here the angeiomatous condition affected only one finger. Also in Mr. Poland's case (*vide infra*), in which a vascular condition affected the dorsum and sole of one foot, ligature of the dorsalis pedis and tibials failed also.

itself and the bluish discoloration were extremely diminished. Elsewhere its former existence was only shown by brownish depressed and very distinctly pigmented scars. Extension of the fingers was complete; flexion was possible to about two-thirds of the full movement only. The fingers were now nearly free from the former feeling of fulness, clumsy bigness, and itching.

While the repeated insertion of catgut setons had had much to do with the great improvement of the disease in the fingers, I was averse to using them in dealing with the swelling which persisted in the palm. In the fingers the setons had been introduced on the back and sides, safely away from the thecæ. But the disease which persisted in the palm lay deeper, at the base of the hypothenar and between this and the thenar eminences, just over the place where the tendons are crowded together. As the setons would have to be introduced deeply here, I preferred to run the risk of tying the radial and ulnar arteries. This was done on August 18th. Feeble pulsation was present in the radial; none in the ulnar. Each stroke of the knife was accompanied by universal, free, punctate oozing; on this account a few threads of horsehair were used for drainage. Several fresh setons of catgut were introduced into the patches of disease still present in the pulps of the index, little, and ring fingers. The hand became cold after the operation, but there was no waxy pallor. The limb was well wrapped up in cotton wool. The two incisions for ligature of the arteries healed without drawback, and no ulceration or threatening of gangrene followed. Henceforward the only treatment employed was firm bandaging of the fingers and hand. After an interval of ten years I am able to give the permanent result of the above operations. They show how complete has been the cure. I quote the account as given by the patient herself and a lady who has been, throughout, much interested in her. Since N. A. left my care I have had no opportunity of seeing her.

"N. A. is now 27. She is head-mistress of a school in North Wales, with an average of forty-five children. After being under your care she was able to pass her three examinations, including at each of the three times a good deal of fine sewing, knitting and

drawing maps. She now teaches sewing, knitting, writing and drawing, herself drawing freely on the black-board for the children. She is in good health and does not suffer from her hand in any way, except that during the winter, cold weather makes it ache."

As angeiomata of the extremities and especially the hand, though happily not common, are extremely difficult to cure radically, the following references to a few cases illustrating, as some of them do, the difficulty sometimes met with in bringing about a radical cure, and the severity of some of the steps taken, may be of interest.

The well-known surgeon of New York, Dr. Abbe, brought before the New York Surgical Society (*Annals of Surgery*, 1894, Vol. i., p. 364), a boy, nine years of age, with an "aneurismal varix" occupying the ring, middle, and index fingers, as well as the palm and dorsum of the right hand. When the patient was a baby a small nævus was first noticed on the dorsum of the middle finger near the middle joint. This remained quiescent many years, but afterwards grew a little larger. About a year ago he sustained a deep cut with a knife at the base of the thumb; soon after, the nævoid degeneration took on a rapid enlargement, until it attained very marked dimensions. The fingers affected have become several times larger than normal. There is a thrill at the base of the thumb, and apparently a connection between the artery and veins at that point, from which point, also, the rapid invasion of the neighbouring parts started. There is undoubtedly some increase in the size of the radial artery all the way up the forearm, but it is likely that this is only coincident with the increased supply of blood demanded by the finger enlargement. There was no question in the family about the rapidity of the growth during the year since the accidental cutting, yet it was not possible to say that there was no connection between the arterial venous plexuses before that time. As there was a very local aneurismal thrill at a point quite near to the scar in this hand, Dr. Abbe believed that there was a connection between a rather large artery and a venous trunk close to the chief enlargement, and therefore preferred to call

the condition one of aneurismal varix rather than angeioma. With regard to treatment, he had operated on one case successfully which resembled the one now before the society. The whole arm was involved from the hand to the shoulder, presenting a sponge-like appearance, but there was not the bruit nor the history of traumatism with more or less rapid increase afterwards. He tried several methods, resorting to excision of a part, to the actual cautery, and cautery with a wire loop, cutting through the masses in planes with the cautery wire. He also tried Roser's method, with the septic setons suggested by Professor Esmareck, who witnessed the cautery operations. But in a case like the present one he would be inclined to cut down upon the point of apparent anastomosis and excise that portion. He intended also to try the Roser method again.

Dr. A. P. Gerster looked upon the case not as one of aneurysm, but of arteriovenous angeioma, a condition very frequently congenital and growing with the growth of the individual, involving not only the vessels, but all the tissues, even the skeleton of the parts affected. He described a similar case, one entire lower extremity up to the buttock being involved. Ultimately the veins became thrombosed, and very severe attacks of phlebitis occurred, accompanied by high fever and serious interference with the circulation of the limb, so that superficial gangrene of the skin developed in several places. In order to ward off a possibly fatal complication which was threatened in this manner, he finally amputated the limb through the diseased tissues. The vascularity of the cut surface was so great that he had, according to the statement of Dr. Kinloch, of Charleston, who assisted him, to tie about seventy-five arteries, yet the surface was still practically a spongy opening, which would have caused fatal hæmorrhage the moment constriction was removed. In order to secure against the possibility of secondary hæmorrhage, he resorted to the plan of closing the entire surface by buried sutures, commencing at the periosteum and going out to the skin. The wound healed by first intention, and the patient was well seven years after the operation. In another case, that of a boy eleven or twelve years of age, the disease being limited to the toes, foot and heel, the

external iliac was first tied, and, as a consequence, rather extensive gangrene of the skin near the disease and of the bellies of both peronæi muscles developed, and the boy's foot had to be amputated. He finally recovered. The bones in both cases were decidedly enlarged, as ascertained by exact measurement.

Other instances of the grave importance which may sometimes be attached to these cases of angeioma of the hand are shown by two of the following cases.

Professor Spence (*Medical Times & Gazette*, vol. ii., 1875, p. 209) related the following cases. "An infant of six weeks was sent to my care on account of a deep-seated erectile tumour occupying the palm of the hand and extending up to the wrist. The tumour had been growing rapidly, and at one point the skin was thin and discoloured. I used injection of perchloride of iron, and part consolidated. Again it was used and the consolidation was followed by inflammation and separation of a small central slough. From the ulcerated surface bleeding took place, and though arrested by local application of the perchloride, it returned from time to time, and as the child's life was thus endangered, and the growth seemed rather to increase than to diminish, I was forced to amputate through the forearm when the infant was eight weeks old. She made a very rapid recovery, but with the loss of a hand. I show you here the cast of the hand of an infant affected with deep-seated pulsating erectile tumour very similar to the former. In this case I applied electrolysis during three months while the child was under my care in hospital, and by several applications of the battery the growth began to consolidate and contract. As it was inconvenient for the mother to remain in hospital, I asked Dr. Connel, of Peebles, who had sent the case to me, to conduct the remainder of the treatment." It is stated that "the cure was completed," but we have no information how long afterwards the case was watched.

The following case is reported by Dr. Russell (*Medical Gazette*, vol. xviii., p. 173). It appears to belong to the second or non-congenital group of these cases. "The woman, æt. 41, had two swellings, each about the size of a walnut, extending one from the extremity of the ring, the other from that of the little

finger to the middle of the fingers, of a violet colour and spongy feel, and of a structure resembling a placenta. They shrunk under pressure, but recovered their size on its being removed; throbbed strongly, as did all the vessels of the arm, and were excessively painful. The radial and ulnar arteries were enlarged, the latter tortuous, the basilic vein had assumed the appearance so commonly seen in a varicose vein of the leg. She stated that the disease had commenced five years before, on the tip of the ring finger, just under the nail, by a bleeding, without any previous symptom, while engaged in wringing clothes. At first she observed a little speck, which ulcerated, cicatrized, and then gradually formed a little violet-coloured pulpy tumour, bleeding frequently and sometimes freely. In three years more a similar tumour appeared on the little finger of the same hand. The bleeding and inconvenience increased much. I recommended removal of the fingers at the metacarpal joints, which she declined. After some weeks she returned, willing to submit to anything. The disease had now extended so far as to form one aneurysmal tumour at the junction of the two fingers, the placenta-like appearance reached now to the wrist, the arm was of an erythematous hue to the elbow, the tumour had ulcerated and sloughed, and the carious phalanges protruded from the gangrenous fingers, which were enlarged and dreadfully painful. The pulsation and size of the arteries and veins had considerably increased up to the axilla. I first tied the ulnar artery at the wrist and then amputated the metacarpal bones of the ring and little fingers at the carpus, being obliged to cut within the verge of the purple mass. The stump healed well. Three years after she called to thank me. All the morbid affections had long subsided, the vessels of the arm had resumed their natural appearance, and with the assistance of the thumb and two fingers she was enabled to follow her occupation of a laundress."

The following case is of interest, (1) from the failure of ligature of the radial ulnar arteries, and (2) from the treatment adopted later. It will be seen that the mischief was probably congenital and affected one finger only. The case is given by Mr. Wardrop

(Med. Chir. Trs., Vol. ix., p. 216). It is related in the words of Sir William Lawrence.

“A woman, twenty-one years old, has been for the last three or four years under the care, first of Mr. Hodgson and subsequently of myself, for a pulsating tumour of the finger, of the description which has been called aneurism by anastomosis. She does not remember its commencement, but rather supposes that it had existed from the time of birth; it increased in size, and began to be troublesome about four years ago. The complaint occupied the ring finger of the right hand, there was a general fulness of the first phalanx, but the chief swelling was on the palmar surface and ulnar side of the finger, the circumference of which may probably have exceeded the natural dimensions by one third. The swelling was soft and compressible; the vessels composing it obscurely discernible through the skin, and gave it a slightly reddish or livid tint. There was a sensation of heat in it, and it was rather warm to the touch. It pulsated strongly, just like an aneurism. The digital artery of the corresponding side was very large, and conspicuous by its size and strong pulsation in the palm of the hand. The veins at the back of the finger, hand, and forearm were turgid, and the integuments of the hand, on its dorsal surface marked by a line of discolouration exactly like that which remains after a bruise. Having ascertained that the beating could be stopped entirely by pressing on the radial and ulnar arteries at the same time, and having tried ineffectually for several months compression and other external means, Mr. Hodgson tied both the trunks mentioned. The consequences of the operation were an entire cessation of the beating, collapse of the swelling and relief of pain, but these symptoms all recurred in a few days, and were just as bad as before.” Compression being again unavailing and amputation of the finger being refused, Sir W. Lawrence operated as follows:—

“To cut off the supply of blood I made a circular cut close to the palm, through all the soft parts, excepting the flexor tendons with their theca, and the extensor tendons. The digital artery, which had pulsated so evidently in the palm of the hand, was fully equal in size to the radial or ulnar of an adult, and was th

principal nutrient vessel of the disease. After tying this and the opposite one we were much surprised at finding so strong a jet of arterial blood from the other orifices of these two vessels as to render ligature necessary. The occurrence, however, dissipated any apprehensions that might have been entertained respecting the subsequent supply of the finger. The edges of the incision were brought together by four sutures, but could not be very satisfactorily united in consequence of the tumor, and indeed, the whole finger beyond the cut swelling considerably. The wound of the incision healed slowly: the swelling subsided, but did not entirely disappear; and the integuments recovered their natural colour. The pulsation and the pain were put an end to. At the present time"—the interval after the operation is not given, but was, perhaps, two years—"there is still a fulness of the part, but without any beating, and some minute red vessels are visible in the skin."

In reporting a case of cavernous angioma of the hand in our Hospital Reports, one naturally refers to a closely similar case of "Erectile Tumour of the Foot," fully reported by Mr. Poland in these Reports for 1868-1869. The wax models, copied from a dissection of the part by Mr. Howse, show the presence in the sole of the foot of cavernous tissue very similar to that in the palm of the hand of my patient.

The patient was a girl of nineteen. The mischief here dated to an injury to the foot from the fall of a desk ten years before. Cavernous tissue was present in this case both in the sole, where it was continuous with the plantar arteries, and in the dorsum where it was connected with the dorsal artery and internal saphenous vein. Mr. Howse who wrote as a supplement to Mr. Poland's article a full account of the anatomy of the growth was inclined to think that it originated in the walls of the arteries and veins, as small growths were found arising in the walls of the digital vessels and the internal saphenous vein quite unconnected with the larger masses of erectile tissue. In this case the dorsal artery was first tied, as pressure on this vessel controlled the pulsation in the cavernous tissue on the dorsum. Pulsation reappearing, the posterior tibial artery was tied in its lower third.

Symptoms completely disappeared, and pressure by a pad and bandage was ordered, the patient leaving the hospital. The swelling on the dorsum reappeared, and as pressure on the anterior tibial just above the ankle commanded this, the artery was tied at the above spot. Once more the swelling, pain, and throbbing disappeared, only to return in three or four weeks. Amputation was now performed through the leg, a good recovery following.

In a similar case of cavernous angeioma of the foot it would be interesting to know the result of first tying the femoral artery and then, if needful, the tibials low down.

CONCLUSIONS.

(i.) The importance of curing early any nævus on the hand or foot.

(ii.) Ligature of the main artery first, and, later on, if needful, of the radial and ulnar or tibial arteries, offers the speediest and simplest method of treatment in these cases, where the arteries, veins and capillaries are all affected, and cavernous tissue is present as well. The above treatment may be supplemented by the use of sterilised setons and firm bandaging, but it is unlikely that these will cure any cavernous tissue which persists after ligature of the main trunk only. Ligature of the radial and ulnar or tibial arteries alone is not likely to bring about a radical cure judging from Mr. Hodgson's case—here only one finger was affected—and that of Mr. Poland.

(iii.) If the criticism is offered that tying the radial and ulnar just above the wrist, after ligature of the brachial, is a dangerous step, I admit its fairness. I would reply that any other treatment of the cavernous tissue which still persisted in the palm, as by setons, excision, or electrolysis, was not promising, owing to the close contiguity of the flexor tendons, together with the nerves and the depth at which the mischief lay. In taking the further step I was influenced by the following points. Five months had elapsed since the ligature of the brachial artery. The patient was young and her vessels free from endarteritis. As it was clear that the anastomoses round the elbow-joint had

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brought sufficient blood into the ulnar and radial arteries to feed the disease which persisted in the palm, it might be expected that, after ligature of the radial and ulnar arteries just above the wrist, sufficient blood would find its way by means of the interosseous and carpal arteries, and those abnormal channels which would be likely to exist in such a case of eighteen years' duration to carry on the life of the hand.

A CASE OF SPLENIC ANÆMIA.

BY LAURISTON E. SHAW, M.D.

PHYSICIAN TO GUY'S HOSPITAL.

IN the fifty-second volume of these Reports Dr. Frederick Taylor has recorded a typical case of Splenic Anæmia. Unfortunately no post-mortem examination could be obtained, but the clinical aspects of the case leave no room for doubt that the disease belonged to the group to which the title splenic anæmia has been given. In the last few years the number of cases recorded as examples of this condition has rapidly increased, and many varying more or less from the type originally described have been included. Osler has collected a series of fifteen cases which occurred in his own practice during twenty years, and has recorded them in the American Journal of the Medical Sciences for January, 1900. In his paper he has drawn attention to the diseases for which this condition is likely to be mistaken, and has given a brief summary of the main clinical features. To this communication, to Dr. Taylor's paper above referred to, and to the article on Splenic Anæmia by Samuel West in Clifford Allbutt's System of Medicine, the reader is referred for particulars as to the nature and history of the disease. In recording the present case some points of special interest will alone be dealt with.

Splenic anæmia has naturally attracted great attention because it appears to be the only uniformly fatal disease in which it is possible that the extirpation of an enlarged spleen may save the

patient's life. In Dr. Taylor's case the possible advantages of an operation were pointed out to the patient's friends, but operation was declined. In the case I am about to record I regret that no operation was proposed. This was not due to a want of appreciation of its possible advantages, but to the uncertainty of the diagnosis. Although the possibility of the case being one of splenic anæmia was before us from the time of the patient's admission to the hospital, and although I had the advantage of consultations with several of my colleagues, I was unable to satisfy myself that the association of enlargement of the spleen with hæmatemesis might not be due to some condition which was recoverable without operation, or was beyond the possibility of relief by surgical means.

I do not wish to be understood as advocating that in all cases of splenic anæmia the spleen should be removed. In any case in which the diagnosis can be confidently made, and in which, notwithstanding medicinal treatment, the disease makes steady progress, splenectomy is justified. In endeavouring to estimate the value of this treatment, two points must be borne in mind. Already a case has been recorded in which good results seemed to follow the operation, but in which a few years later death ensued, and the post-mortem examination showed the enlargement of the spleen to have been due to cirrhosis of the liver. At present, also, our knowledge of the life-history of the disease is very imperfect. From some of Osler's recently reported cases it appears that the disease may exist much longer than the period of two years, which was until lately stated to be its maximum duration. Even should a patient survive splenectomy for twelve years, we cannot say for certain that this is in consequence of, and not despite the operation. It must not be forgotten that at one time splenectomy was regarded as a proper proceeding in leucocythæmia. It is now altogether abandoned, on account of the serious risk of uncontrollable hæmorrhage, due to the altered state of the blood. Neither in leucocythæmia nor in splenic anæmia have we any definite knowledge as to the actual cause of the disease, nor can we say for certain whether the enlarged spleen is a primary or secondary factor in the pathology. Until

our knowledge is greater our treatment must be empirical. Experience has proved that no immediate ill effects follow the removal of the spleen in splenic anæmia. Extended knowledge will shew whether the good effects are permanent, and whether simpler measures can be relied upon to produce equally good results.

The following notes are from the report written by Mr. H. M. Hardy :—

Lily D., æt. 10 years, was admitted into Guy's Hospital on January 19th, 1898, for a tumour in the abdomen.

Family history.—The father and mother are quite healthy. The father is a bricklayer's labourer. Two brothers died in infancy, the remainder of the family, three in number, are healthy.

Previous history.—The child has bronchitis every winter. She has had measles, whooping cough, and chicken-pox. Has always been considered by her mother to be a weakly child, often complaining of headache. She has always lived at Greenhithe, in Kent.

History of present illness.—On December 26th, 1897, viz., twenty-four days before admission, while in her usual health the patient awoke at 4 a.m. and called out that she was going to be sick. She immediately vomited half-a-pint of blood mixed with food. The vomiting of blood was repeated four times on the 26th of December, and twice on the 27th. The medical man who was called in to see the child noticed a tumour in the abdomen. Since the attack of hæmatemesis the patient has been pale and has lost flesh. There has been no acute pain, and no further gastric disturbance.

Condition on admission.—The patient is anæmic, the skin having a yellowish tinge. She is fairly well nourished. The temperature is 99°, the respirations are 28 per minute; there is no cough or dyspnoea, and the physical examination of the lungs reveals no abnormality.

The pulse is 100, small, regular, and compressible. There is a soft blowing systolic murmur at the apex, but no obvious enlargement of the heart. The apex beat is in the fourth space.

The specific gravity of the urine is 1012, its reaction neutral, there are no abnormal constituents.

The lips are pale, the tongue moist, the appetite is good; the bowels are regular.

On examination of the abdomen, the spleen is found to be much enlarged, forming a tumour reaching from the costal margin to the left iliac fossa. Its inner edge is sharp and well defined, and close to the costal margin a notch can be felt. The tumour moves freely on respiration.

The liver dulness is not increased, but the edge of the organ can be felt to descend below the costal margin on deep inspiration and is perhaps a little harder than normal.

The abdomen appears to be distended and the veins over it are unnaturally visible. No ascites can be detected.

Examination of blood.—Hæmoglobin, 80 per cent.; red corpuscles, 3,300,000 per cubic millimetre; white corpuscles, no increase.

The patient was put on *Mist. Ferri Tartarati* $\mathfrak{z}\text{ij}$. *ter die*, with farinaceous diet and beef-tea.

January 31st. The edge of the liver is clearly felt just below the costal margin and is thought to be a little harder than normal. There is some increase in the amount of abdominal distension. The hæmoglobin has increased to 45 per cent.

February 2nd. The backs of the eyes appear quite normal. No hæmorrhage can be detected.

3rd. The blood is examined to-day and gives the following results:—Red discs, 3,000,000; hæmoglobin, 60 per cent.; white corpuscles, two or three only seen in the field.

6th. Temperature 102°. Slight epistaxis this morning. Patient complaining of headache and some pains in the joints. The medicine has been changed to a mixture containing two drops of *Liq. Arsenicalis*, and two grains of *Ferri et Am. Cit.* each dose. The patient is on fish diet.

14th. The spleen is harder but not materially altered in size. Hæmoglobin, 50 per cent.; red corpuscles, 4,000,000; white corpuscles, no excess. Circumference of the abdomen at the level of umbilicus, twenty-three inches.

15th. Patient to-day vomited about a pint of blood, at first bright red and later dark in colour. A few hours after the hæmatemesis the spleen was noticed to be very much diminished in size.

17th. The patient vomited half a pint of blood yesterday and a smaller quantity to-day. The spleen is still more diminished in size.

18th. No more hæmatemesis. The circumference of the abdomen is twenty-one and three-eighths inches, being one and five-eighths inches less than it was the day before the recent attack of hæmatemesis. Since the vomiting the patient has been fed on peptonised milk and nutrient enemata.

23rd. The spleen is larger again. It reaches from the seventh rib to within an inch of the anterior superior spine and forwards to within two inches of the umbilicus.

March 1st. The abdomen is more distended. It measures twenty-three inches.

5th. Blood: red corpuscles, 2,850,000 per cubic millimetre; hæmoglobin, 30 per cent.; white corpuscles 1 to 912 red.

11th. Another attack of hæmatemesis. Vomiting at 4 p.m., again at 11 p.m. Altogether two pints of fluid were brought up, chiefly consisting of blood.

12th. To-day two or three ounces of blood were vomited in the morning, and more than a pint in the afternoon. The circumference of the abdomen is twenty and three-quarter inches. The liver and spleen both seem diminished in size.

14th. No more hæmatemesis. Patient is looking very anæmic. There is a systolic bruit at the apex and another of different character over the pulmonary area. Abdomen twenty and a half inches.

25th. Urine 1020, normal. Abdomen much distended, circumference twenty-four and a half inches. Some dulness in right flank which disappears on rolling patient on to the left side. No thrill. Liver is felt well below the costal margin.

28th. Slight epistaxis this morning.

April 5th. Red corpuscles, 4,250,000; hæmoglobin, 45 per cent.; white corpuscles, no excess.

13th. Spleen seems larger and is very hard, it reaches within a finger's breadth of the crest of the ilium.

May 5th. Patient vomited several times to-day. The vomited matter is blood-stained. The spleen feels softer.

6th. Slight recurrence of hæmatemesis.

14th. Patient looks very pale and ill. The abdomen is generally tender, especially so over the spleen.

15th. There is a small quantity of dark brown vomit to-day.

20th. She looks brighter, but is absolutely colourless.

June 6th. There is much pain in the abdomen, aggravated by coughing.

10th. In great pain, frequently sick, no hæmatemesis.

13th. Constant sickness. Bowels open nine times in the last twenty-four hours. Some of the motions contain blood.

14th. The patient became collapsed and died this morning.

The post-mortem examination was made by Dr. Perry. The following abstract is from his report of the case:—

The mucous membrane of the stomach was pale. No ulcer or other source of hæmorrhage was detected.

The spleen weighed fourteen ounces, and on its surface were numerous small tufts resembling the vascular tufts seen on the surface of the liver in chronic venous engorgement. On section, the structure of the organ appeared normal.

The liver was little, if at all enlarged; it was pale on section, and appeared normal but for some tough adhesions around the gall-bladder.

The heart was normal, weighing four and a-half ounces.

Recent ante-mortem thrombus was found in several vessels. Just at the commencement of the right branch of the pulmonary artery there was a small ante-mortem thrombus, about the size of a pea, reddish grey in colour, and adherent to the wall of the vessel.

The main trunk of the portal vein was found plugged with recent thrombus, which did not extend into the branches of the portal vein in the liver. The veins of the stomach were not thrombosed. The superior mesenteric vein was plugged, and there was some thrombosis of the terminal portion of the inferior

mesenteric. At the lower part of the small intestine there was an area of intense congestion, the part affected being the last eight inches or so of the ileum. The upper limit of the congestion was well marked, while the lower was less well defined. On the surface of the congested bowel was a thin deposit of lymph, and the peritoneal cavity contained an ounce or two of bloody fluid. On gently squeezing the intestine some fæcal exudation took place through several small apertures. The mucous membrane at this part was of a dark red colour, granular on the surface, and evidently undergoing coagulation necrosis. Tracing up the superior mesenteric vein from this part it was found to be plugged throughout, and the thrombus was continuous with the thrombus in the portal vein. The main splenic vein was plugged by somewhat adherent ante-mortem thrombus. This thrombus was only about an inch in length, and did not appear to be continuous with that in the portal vein.

The thrombosis in all the vessels was obviously of very recent date.

The rest of the viscera examined were normal.

I have recorded this case at some length as one of splenic anæmia, because it seems desirable that we should carefully compare all cases of enlarged spleen of obscure origin associated with anæmia, in order to determine whether there is a group of cases sufficiently defined to warrant us in giving them a special name and searching for a common cause. It is probable that, owing to the interest lately aroused in this subject, and to the satisfaction usually experienced in making a definite diagnosis, most obscure diseases associated with splenic enlargement will be classified as "splenic anæmia." This will ensure such cases being carefully contrasted, and will assist the task of subdivision, which, I believe will soon be found necessary. As long as we can, we force anomalous cases into existing groups of well-established diseases, making light of the features in which they fail to conform to the type. As soon as a new disease is "discovered," the anomalous cases, which have with difficulty been retained in a large number of quite dissimilar groups, are transferred to this new place. Thus it is the number of recorded cases

of splenic anæmia is rapidly increasing, and the dissimilarity of many of the cases is very striking.

I am by no means certain that my case would be accepted as an instance of splenic anæmia by all those who have written on the subject or have recorded cases. If it is not, however, I do not know under what other well-established disease it should be classified.

It certainly was not a case of leucocythæmia, nor cirrhosis of liver, nor malarial cachexia. While the case was under my care I regarded it as one of portal obstruction, attributing the enlargement of the spleen and the hæmatemesis to the results of impeded circulation, and the anæmia to the repeated loss of blood. I thought it possible that we should find some organic cause for such portal obstruction in cirrhosis of the liver, in thrombosis of veins, or in permanent obstruction by enlarged glands or by condensed fibrous tissue. As already stated, the question of splenic anæmia was fully considered at the bedside, but no definite opinion, either for or against the diagnosis was expressed. The child did not come under observation till it had suffered from profuse hæmatemesis, and there was no evidence that it had suffered from anæmia until after it had lost a large amount of blood. A feature of the case which, to my mind, pointed to portal obstruction, was the diminution in the size of the spleen which followed the various attacks of hæmatemesis and the subsequent progressive swelling of the abdomen, which was obviously not due to, although it kept pace with, the enlargement of the spleen.

The post-mortem proved the absence of any cause of portal obstruction, and practically established the case as one of splenic anæmia.

But there is a strong suspicion in my mind that, as is common whenever a new disease is "discovered," we are forcing into a single group cases which further knowledge will lead us to differentiate.

Samuel West, writing in 1898, and analysing the symptoms of the cases recorded up to that date makes the following statement:—

“ Hæmorrhages.—The tendency to bleeding is pronounced ; the hæmorrhages are usually of slight degree and of the nature of oozing ; but they frequently recur, are very difficult to control, and add greatly to the anæmia. * * * * *From the gastrointestinal organs hæmorrhage is rare, and if any large amount it is probably associated with some secondary lesion.* A case is, however, recorded by Dr. Douglas Stanley, in which, although profuse and fatal hæmatemesis took place, no lesion in the stomach was found after death.”

In Osler's collected cases, on the other hand, profuse hæmatemesis is a very common symptom. Out of a total of fifteen cases, eight suffered more or less from hæmatemesis, and in several cases bleeding occurred from no other organ than the stomach, and in some as in this case I am now recording, the recurrent hæmatemesis appears to have been the direct cause of death.

The question that I desire to raise, and which indeed seems forced upon us by the dissimilarity of Brühl's and Osler's cases, is whether we are not dealing with two quite separate groups ? In one group the course of events appears to be (1) large spleen, (2) anæmia, (3) general hæmorrhagic tendency ; in the other, (1) large spleen, (2) local gastric hæmorrhage, (3) anæmia.

Douglas Stanley's case, many of Osler's cases, and the case now recorded, should be placed in the second group. Most of Brühl's cases and Dr. Taylor's case belong to the first group. In estimating the value of splenectomy in the treatment of splenic anæmia, it must be carefully considered to which of these groups the case belongs. The hope of good being done by the operation should certainly be greater in cases belonging to the second group.

SOME CONDITIONS IN WHICH OPIUM IS DANGEROUS.

BY A. P. BEDDARD, M.D.
SENIOR DEMONSTRATOR OF PHYSIOLOGY.

It happens occasionally that a patient dies so soon after an ordinary dose of opium—and this applies especially to hypodermic injections of morphia—as to leave but little doubt that the opium has been the immediate cause of death. It seems of some practical importance, therefore, to enquire what class of patients are thus liable to be killed by a small dose of opium, and how the opium brings about rapid death.

On turning to Allbutt's System of Medicine, as the largest English text-book, for guidance in giving opium in diseases of the kidneys, heart and lungs, the following statements bearing on the subject are found:—"Opium and its derivatives should be rigidly avoided in the convulsive and every other stage of organic albuminuria, save only with the lardaceous kidney, where they are permissible and sometimes useful, but not when this condition is productive of convulsion or other uræmic symptom."

In the article on acute pericarditis, only two statements are made:—"Should the pain be severe, opium may be given, Dover's powder being a useful preparation; or morphia may be administered subcutaneously and repeated as occasion demands." And in speaking of severe cases with dyspnœa, cyanosis and want of sleep:—"Subcutaneous injection of morphine may be imperatively demanded, even if risky. Dr. Cheadle speaks highly of nepenthe for children." The article on disease of the

aortic area of the heart mentions generally the value of the hypodermic injection of morphine in heart disease, quotes Leonard Hill to the effect that "morphine is one of the best vaso-constrictors and cardiac tonics we possess," and gives the following warnings:—"Of the drawbacks to the continuous use of morphine I may refer to the article on the subject. Like any other potent remedy it must be used seasonably and discreetly." The article on diseases of the mitral valve mentions that "In a considerable number of cases manifesting distressful symptoms of dyspnoea and insomnia no agent succeeds so well as morphia. By far the best way of administering it in cases of cardiac disease is by hypodermic injection. The first dose should be small—one-sixth or one-fourth of a grain—but this may be increased subsequently to half a grain. Care should be taken that the administration shall not become habitual." The article on angina pectoris, after speaking of the use of the nitrites, goes on to say:—"When pain is not relieved by this treatment, arterial spasm having been eliminated as its cause, the use of subcutaneous morphia is indicated, due care being exercised with regard to the dose in view of the possible presence of kidney disease; if this factor be excluded, the degree of pain would regulate the dose and the combination of atropine would be useful as a heart stimulant. The free use of oxygen inhalation is of very great value in all cases in which cardiac failure is a marked feature. . . . Oxygen inhalation is particularly indicated in those cases in which morphia is found necessary; and when the paroxysm is over and sleep induced, the gas should be allowed from time to time to fortify the air immediately above the patient's mouth and nose."

In the treatment of pneumonia it is stated that for severe sleeplessness opium "has been forbidden lest its use should increase cyanosis, diminish respiratory efforts and lead to fatal coma. These fears are far from groundless. When there is extensive consolidation or much bronchitis, when the patient is livid, and the expectoration scanty, it would be bad practice to give this drug." And further on, "In many cases of pneumonia the danger is not directly from suffocation, it is rather from the

effects of a continued high temperature upon the heart, the impending weakness of the respiratory muscles and the exhaustion of the reflex activity of the nervous centres. In such cases ten grains of Dover's powder, five grains of compound soap pill, fifteen to twenty drops of laudanum are often invaluable and succeed when all other hypnotics are powerless. An additional warrant for the use of opium is dilatation of the pupils. The presence of albumen, if only of pyrexial origin, is no counter-indication; but if the patient be a subject of chronic Bright's disease we must forego the use of this valuable drug, or use it at his peril, to escape a still more imminent danger." Again, in the treatment of attacks of spasmodic dyspnoea in emphysema with bronchitis it is stated that "The desirability of employing morphia in such cases will depend chiefly on the relative preponderance of the spasmodic or catarrhal factor. The nearer the attack approaches in character to one of true asthma, the greater is the probability of relief from a subcutaneous injection of morphia; whilst, on the other hand, if the dyspnoea be chiefly due to the accompanying bronchitis, the use of morphia may be attended with the greatest danger."

The advice contained in the above extracts may be briefly and fairly summarised as follows:—Opium in albuminuria due to nephritis is so dangerous as to be inadmissible, presumably, although it is not so stated, because the disease of the kidneys will interfere with the elimination of the morphia in the urine. In cardiac failure due to various diseases of the heart, opium is in some cases excellent treatment, in others it is less safe, although in what the danger really consists and how it may be clinically recognised is not very precisely stated. In acute inflammation of the lungs opium may be highly dangerous, but here again the exact nature and cause of the danger is not made very clear.

When, however, we turn to the known physiological actions of opium and its method of elimination from the body as the basis on which to guide us in the use of it in disease, it becomes doubtful whether much of the above quoted advice can really be founded on facts. However opium is introduced into the body, whether by the alimentary canal or subcutaneously, it or its

products of oxidation in the body are ultimately eliminated from the blood almost entirely by the mucous membrane of the stomach and intestines. Minute quantities of it do leave by the kidneys, but the most reliable work on the subject shows them to be insignificant. This at once raises a doubt as to whether opium must of necessity be dangerous in all cases of organic albuminuria; this doubt is further strengthened by the following considerations: Cases of acute uræmic convulsions, of uræmic dyspnoea and of puerperal eclampsia are not infrequently now treated by hypodermic injections of morphia, and treated with success. If opium were really dangerous in renal disease simply because of its non-excretion in the urine, then it ought to be equally dangerous in any condition in which the secretion of urine is very slight, as in shock, acute peritonitis, acute intestinal obstruction and collapse generally, also in the backward venous pressure of cardiac and pulmonary disease, in all of which the secretion of urine may be as nearly suppressed as in nephritis; yet in many of these conditions opium is not considered an unsafe remedy. Again, many of the fatal cases are not of patients suffering with organic renal disease at all, and simply because a patient passing albuminous urine dies after a dose of opium, it does not follow that death is caused by the kidneys failing to excrete the morphia.

Turning now to the physiological actions of opium, we find that besides its depressing action on the higher parts of the central nervous system, whereby it relieves pain, its chief well recognised effects are—(1) its special depressing action on the cells of the respiratory centre, and (2) its action on the heart as a powerful cardiac stimulant and on the vasomotor centre, causing vasoconstriction of the peripheral arterioles. It is by a careful consideration of these two effects that we find an explanation of the cases under consideration and also the main indications of when and when not opium is dangerous.

It is a mistake to think that even in health the contraction of the heart and the elastic recoil of the larger arteries are the only forces used to send the blood round the circulation. The so-called respiratory pump is more or less necessary, we will

therefore consider its mode of action. In man, in the erect posture, the chief problem of the venous circulation is how to get the blood from the legs and abdomen back to the heart against the force of gravity; when in the horizontal position, although the action of gravity is eliminated, a proper return of blood from the abdomen is still a matter of prime importance to the circulation and requires a special mechanism to ensure it. The return of blood from the legs is largely carried out by the compression of the deep veins by the limb muscles, and though important, its importance is slight as compared with the question of the return of blood from the abdomen, the readily distensible veins of which could hold the total circulating blood. A properly maintained abdominal circulation is therefore of great importance to the whole body, and especially to the brain. It is controlled by two mechanisms, one to prevent an undue quantity of blood from ever reaching the abdominal veins, namely, the vasomotor centre and nerves keeping the splanchnic arterioles in various degrees of constriction, and a second to actively empty the abdominal veins, namely, the respiratory pump.

This pumping action of the respiratory movements combines the two actions both of a suction pump and of a force pump. The suction action takes place only during inspiration and is produced by the increased negative pressure in the thorax actively sucking blood from the abdomen into the thorax. In the absence of exertion during health, that is with a strong heart, elastic arteries and a properly acting vasomotor centre, this suction adequately empties the abdominal veins and fills the right heart. The force-pump action of the respiratory movements is more complicated, but can be much more powerful. The abdomen is a muscular box completely filled by viscera and blood-vessels, and since the venous blood is the only content which can rapidly vary in quantity, it follows that the firmness or laxity of the abdominal walls will determine the size of the abdominal veins, and, therefore, the quantity of blood they contain at any given time. When the diaphragm descends in inspiration the abdominal viscera and veins are compressed and venous blood is forced out of the abdomen into the right

ventricle. This, however, can only take place provided that the muscles of the abdominal wall, under control of the respiratory centre, are capable of resisting the increased intra-abdominal pressure and do not materially stretch. Consequently we find that in some forms of dyspnœa and in muscular exertion, at the same time that the diaphragm descends, the abdominal walls are not only not allowed to give at all, but are actively contracted in order to ensure this pumping action. The power of this force-pump is great, and when in vigorous action it would inevitably overstretch the right ventricle were the latter not supported by the tough inelastic pericardium. In urgent dyspnœa of circulatory origin this pump is capable of driving blood straight through the distended right ventricle into the lungs; and during expiration the lungs are compressed and the blood driven onwards into the left ventricle. Thus by itself the respiratory force pump can to a large extent carry on the circulation apart from the heart, but as has been already explained, in health, this force-pump is only used during exertion and in disease either to supplement the pumping action of the right ventricle or under special circumstances to fill it as well. The respiratory suction-pump is, of course, always at work to a greater or less extent—to a less extent in the horizontal position and to a greater extent in the foot down position. But even in the horizontal position and with a healthy heart it is probable that the circulation as a whole could not be maintained for long without the action of the respiratory pump.

In order to understand the relation that the respiratory pump has to circulatory failure, it is necessary to consider the effect that circulatory failure has upon the respiratory centre. This centre by the respiratory movements maintains a proper interchange of gases in the lungs and also helps to keep up a proper circulation throughout the lungs, we therefore find that it is wonderfully sensitive, not only to the quality but also to the quantity—or rather the velocity of flow—of the blood it revives. The circulation may fail, either on the arterial or venous side, or both, and it makes a considerable difference to the respiratory centre how it fails.

On the arterial side the circulation fails either from failure of the vasomotor centre, as in shock, or from failure of the left ventricle, as in the early stages of uncompensated aortic disease. In both cases the blood is properly aerated, but there is an exaggeration in rate or amplitude of the respiratory movements due to the low aortic blood-pressure and the consequent less rapid flow of blood through the medulla. With regard to this dyspnoea, we have to ask ourselves, Is it of any vital importance for carrying on the circulation and is it therefore any contra-indication to the use of opium? In both cases the dyspnoea is of the suction-pump type, and its object seems to be to hurry blood from the venous system into the right heart and to get it pumped into the left side in order to raise the arterial blood-pressure and so improve the cerebral circulation. In shock and other similar conditions the most effective way of raising the arterial blood-pressure is to replace the action of the vasomotor centre by a firm bandage round the abdomen, and if this be done the dyspnoea will either cease or at least be not in any way essential to the circulation. In such a case morphia is not only not contra-indicated, but may do good both by depressing the brain and rendering it less susceptible to afferent inhibitory impulses, but also, by causing peripheral vaso-constriction, and by stimulating the healthy and in this case underworked cardiac muscle.

In aortic disease with comparative failure of the left ventricle alone, the dyspnoea again is simply the expression of the effect that a low mean general arterial blood-pressure has upon the respiratory centre. The dyspnoea is undoubtedly an attempt to improve the circulation through the medulla on its arterial side, but it is not, as such, essential to the circulation as a whole. Consequently it is not only safe to reduce dyspnoea and subjective distress by opium, but the opium, by its action on the heart and blood-vessels, will improve the circulation on its arterial side and steady the heart.

When we come to cases of failure of the right ventricle we find a totally different circulatory condition. Whenever the muscle of the right ventricle begins to fail from overwork, however caused, it dilates, the residual blood in it is increased, and the more it

dilates the more work the muscle has to do to send out an adequate quantity of blood. Therefore, a vicious circle is easily set up, the arterial blood-pressure falls, whilst that in the veins rises and the velocity in the pulmonary, coronary, and especially the cerebral circulations, is greatly slowed. The whole circulation would run a great risk of coming to a standstill unless the respiratory pump came to the assistance of the right ventricle. The respiratory centre is stimulated partly by the less well aerated blood supplied to it, and partly by the less rapid flow of blood through the whole brain. It has already been pointed out that the respiratory force-pump is of great power, and at first sight it might seem a doubtful benefit to pump more blood into the already over-dilated and labouring right ventricle. This difficulty, however, is only apparent, and is overcome in two ways. In the first place, by the support of the tough inelastic pericardium, which is able to withstand a pressure of nearly two atmospheres and limits the capacity of the heart by about one-half, so that blood forced into the right ventricle under these circumstances can not simply distend it further, but must force blood out of the ventricle into the lungs; and in the second place, by the position of orthopnoea which the patient assumes. By allowing the force of gravity to act on the column of blood in his inferior vena cava, he practically bleeds himself into his abdominal veins, and by the action of his respiratory pump he drives or sucks appropriate quantities of blood through his right ventricle into the lungs. It is practically true to say that whenever a patient with right-sided failure assumes this position his right side is engorged and would be benefited by venesection. And further, the degree in which his respiratory pump is really carrying on the circulation may be gauged by observing the degree in which the abdominal muscles are contracted during inspiration, and the lungs are emptied into the left ventricle by an expiratory dyspnoea.

It is almost unnecessary to point out that whenever the maintenance of the circulation depends materially, as it does in this case, upon the action of the respiratory pump, opium, by its specific action on the respiratory centre may be a dangerous

drug. Of course it is and may be given time after time with impunity, and even with benefit to the patient; here we are only concerned in pointing out in what the danger consists and that the margin of safety is not great and extremely difficult to estimate clinically.

It is in cases of pericarditis with effusion that the most powerful and urgent use of the respiratory force-pump is seen. During inspiration blood has to be driven through the compressed right auricle on into the ventricle in order to fill it, and during expiration the lungs have to be compressed in order to drive blood through the strangled left auricle. In most cases of general circulatory disturbance due to heart failure the dyspnoea is wholly compensatory and beneficial, but in pericarditis with or without myocarditis, or in pericarditis with effusion, there is another side to the question. The heart is now only supported by a softened and distensible pericardium, and the force of the respiratory pump, although necessary to carry on the circulation, at the same time stretches the pericardium and dilates the heart, it may be acutely and irreparably. It may be necessary and even advisable to give opium in these cases but it is certainly a dangerous drug and must be treated as such.

In almost any case of angina pectoris morphia may be safely given during the acute attack, not because these attacks may not be accompanied by considerable cardiac and general circulatory failure, but because the pain itself to a large extent prevents any depressant action of opium on the respiratory centre.

In acute inflammations of the lungs diminished aeration of the blood is a possible cause of severe dyspnoea. This may be present alone or combined with right-sided failure, and the difference between the two cases is well seen in the way they respond to treatment by inhalations of oxygen. If the right ventricle be acting well and the cause of the dyspnoea be want of proper aeration of the blood, oxygen inhalations may for a time improve the dyspnoea; if, however, the right ventricle has failed as well, the inhalations will appear to produce but little effect on the dyspnoea. But even in this latter case inhalations of oxygen do real good because, quite apart from the benefit the cardiac

muscle will derive from a supply of better blood, the safety of the whole circulation lies in the very fact that the respiratory centre normally is and should be kept as susceptible as possible to changes in the quality and quantity of blood supplied to it. It is serious enough that the centre may become gradually narcotised by CO_2 , but to dull it further by opium may be to remove the only effectual defensive mechanism the patient has. As Sir Thomas Watson well says, "Opium is a ticklish remedy in these cases. Many a patient—some within my own knowledge—labouring under general or extensive bronchitis, have been put so soundly to sleep by a dose of opium on going to bed that they have not waked again." It is a familiar fact that but few patients showing Cheyne-Stokes' respiration, associated with right sided failure, recover. This is only another way of saying that by the time the respiratory centre is so exhausted or poisoned that it can only respond with an intermittent activity, the circulation is in imminent danger of stopping.

It has been frequently pointed out that in acute pulmonary disease the size of the pupils may be used to indicate whether or not opium may be safely given. In the quotation given above it is stated that in certain stages of pneumonia "an additional warrant for the use of opium is dilatation of the pupils." It would perhaps be even better to say that constriction of the pupils is an additional contra-indication to its use. In the early stages of disturbance of the cerebral circulation and of CO_2 poisoning the pupils gradually constrict, and by the time they are markedly constricted, the patient is dull and takes but little notice of what goes on around him; in the final stages of poisoning the pupils dilate again and the patient becomes more or less unconscious. In the early stages the respiratory centre is stimulated and the patient sits up in bed and gives his respiratory pump full play; in the later stages the centre becomes poisoned, exhausted and less regularly active, and the patient sinks down in bed, like his centre, unable to respond further to calls upon him. The size of the pupils, therefore, is some indication of the amount of risk the respiratory centre will run during the next few hours of being poisoned. If the pupils are already small, opium is

contra-indicated, but if they are not, from that one observation alone it would be unsafe to infer that opium might be given without risk. In fact, in pulmonary disease, just as in cardiac disease, before opium is given, in any case the question that has to be decided is to what extent the respiratory centre is by its present activity carrying on the circulation, and therefore to what extent is it safe to reduce its excitability by opium.

In organic albuminuria, we have seen that it is hard to believe that opium is necessarily dangerous, and yet such a widespread belief must have some foundation on fact. On the other hand, various considerations have led us to believe that opium may be dangerous in serious right-sided failure with or without albuminuria. Considered from the point of view of their circulation, all cases of nephritis may be divided into two groups—(1) those with an arterial blood-pressure decidedly above the normal—apart, of course, from mere thickening or rigidity of the arterial wall—and (2) those with an arterial pressure that is not above the normal or even distinctly below it. To deal with group (1), which includes the minority of the cases which we have to treat. A high arterial blood-pressure is to be expected in all cases of nephritis, and yet it is seen rarely in cases of chronic nephritis when they come under treatment, more frequently in cases of genuine acute nephritis, sometimes in uræmia, and more often in eclampsia. This rise in arterial blood-pressure is only possible provided the cardiac muscle is acting forcibly and well; and as long as this continues to be the case, opium might be safely given. But it is necessary to remember that a really high arterial blood-pressure, when prolonged, greatly increases the work of the left ventricle, and may so rapidly do so as to cause even acute dilatation of the heart and general circulatory failure; and that simply because a case is one of acute nephritis, for instance, and has a high arterial blood-pressure, it does not follow that it will continue to have it for long; that convulsions, like severe cough, throw an enormous strain on the right ventricle, and of themselves cause a general circulatory disturbance, lowering arterial and raising the venous blood-pressures. Consequently a heart may begin to fail at any moment, and fail

rapidly, letting down the arterial blood-pressure as it fails. This is true not only of uræmic, but of any convulsions; thus it is a well recognised fact that even a small dose of morphia may be extremely dangerous and even fatal in status epilepticus. Further, it must be remembered that in, at any rate, uræmia and eclampsia there is circulating in the blood one or more poisons of whose properties we know little, except that they affect especially the cells of the central nervous system, and as in the case of CO_2 poisoning this might well make us careful of adding another depressant like opium. As in the case of CO_2 poisoning, too, the size of the pupils is of some help to us, for smallness of the pupils contra-indicates the giving of opium. It is impossible to say whether this constriction of the pupils in uræmia is due to a special poison or simply to the less perfect aeration of the blood and disturbance of the cerebral circulation, or to both, but in any case its significance is much the same.

There is one condition, namely, uræmic dyspnœa, in which morphia has been found of great value. The type of respiration is a hyperpnœa, *i.e.* a mere exaggeration in rate and amplitude of the normal respiratory pump, and as such it does the circulation nothing but good. This dyspnœa is not due to circulatory failure nor to imperfect aeration of the blood but is of central origin and presumably due to the action of some poison on the respiratory centre. It frequently occurs as an early and it may be the sole symptoms of uræmia, and provided the heart is working well there could be no objection to treating it by morphia.

Group (2) contains the bulk of the cases of nephritis when they come before us for treatment. Presumably their arterial blood-pressure was once above normal, and that it is no longer so may be due to their nephritis becoming cured, but most frequently it is due to some degree of cardiac failure, much more rarely to collapse. Putting the cases due to collapse aside—and they are readily recognisable—it may be broadly said that opium may be highly dangerous in any case or stage of nephritis in which the arterial blood-pressure is markedly below the normal; just as it may be in cardiac and pulmonary disease and for the same reasons.

The conclusion has thus been reached that the danger of opium in all these cases is due to its depressing action on the respiratory centre, whereby the centre either gives less assistance to a failing circulation or is made too dull to respond to a call for further help. And it must be noted that these remarks apply with even greater force to doses of opium given at night, a time when right-sided circulatory failure is nearly always aggravated and the activity of the central nervous system depressed.

It is impossible here to discuss further the points on which we have to rely clinically for estimating in any given patient the degree in which the respiratory centre is carrying on the circulation. But it must be pointed out that lividity means more than imperfect aeration of the blood, for the total cessation of respiration may not cause any cyanosis. This condition is frequently seen in cases of severe intracranial pressure in which the heart may go on beating for half an hour after respiration has stopped. This means that the blood, although excessively venous, has accumulated, not in the superficial veins at all but almost entirely in the great abdominal veins; and it has done so, because both the mechanisms for keeping the blood from stagnating in the abdominal veins, namely, the vasomotor centre and the respiratory pump, have ceased to act. When, therefore, we see lividity we know that the vasomotor centre is still working, for if its activity decreased the cyanosis would also begin to disappear.

There is still the question of the smallness of the dose which is sometimes fatal, and here failure of elimination, not by the kidneys, but by the alimentary canal, probably plays an important part. In a healthy individual morphia begins to be excreted into the stomach two and a half minutes after a hypodermic injection. Nothing is known as to the effect that right sided cardiac failure has upon this excretion, but judging from the fact that right sided failure means stagnation of blood in the abdominal veins, and that this backward pressure often has a marked effect upon the secretory activities of the alimentary canal, it is hard to believe that the excretion of morphia will in such cases be as rapid as in health. It is this derangement of the circulation through the alimentary canal which probably explains the occasional serious effect of a small dose of opium in cases of cirrhosis of the liver.

It is necessary to say a word about the action of opium on the heart and blood-vessels. The exact action on the heart is that it causes the cardiac muscle to dilate more during diastole and contract up more during systole, and hence it increases largely the amount of blood sent out at each systole. It appears, therefore, to be a direct stimulant to the cardiac muscle and renders a slightly irregular heart regular again. It also contracts the peripheral arterioles by stimulating the vasomotor centre; this chiefly affects the splanchnic area, and the effect here may be so great, that the arterioles of the skin are forced open and dilated.

In a physiological experiment in which powerful respiratory movements are kept going by mechanical means at a certain rate, no matter what effect the opium may have on the animal's respiratory centre, the total effect of opium on the circulation is very like that of digitalis. But in man the conditions are different. Here the total effect of opium on the circulation is the difference of two opposite actions—(1) its power to raise the arterial and lower the venous blood-pressure by increasing the systolic output of the heart and causing splanchnic vaso-constriction, and (2) its power to decrease the pumping of blood from the abdominal veins into the heart, and so raise the venous and lower the arterial blood-pressure. There are, therefore, three possible results, either of these two opposite effects may preponderate or they may mutually counteract each other and leave the circulation unaffected. There are some circulatory disturbances, such as shock, and some cases of aortic disease in which opium may be given with safety; there are others, such as severe pericarditis and pericardial effusion, in which the respiratory pump may be carrying on the circulation almost single-handed and in which opium must be highly dangerous; there are many more in which the respiratory pump is helping an overworked right side, and in which no one can prophecy with absolute certainty what final effect even a small dose of opium will have on the circulation as a whole, and herein lies its danger. In a doubtful case it is safer to give morphia by the mouth than hypodermically, and if given hypodermically, it would make it safer to add atropine or caffein to the injection and give the patient inhalations of oxygen.

A RESEARCH UPON THE NITROGENOUS METABOLISM IN A CASE OF BRIGHT'S DISEASE.

BY J. A. BUTLER, M.B., B.S. LOND., AND H. S. FRENCH,
B.A., B.M., B.CH. OXON.

(From the Physiological Laboratory, Guy's Hospital.)

INTRODUCTION.

(The Figures after Observers' Names indicate the Numbers of
the References given at the end of the Paper).

As far as we have been able to discover, the only published experiments upon the Nitrogenous Metabolism in Bright's disease have been continuous for but short periods. The longest we have found is of twelve days' duration, upon a man aged 47, with chronic interstitial nephritis, by Prior (50) in 1891. The uric acid, the total urinary nitrogen, and the fæces nitrogen were estimated; the food nitrogen was sometimes estimated, and sometimes calculated from diet tables, whilst the urea was not estimated at all. Upon two other occasions Prior (50) estimated the uric acid, the albumen, the urea, the total urinary nitrogen and the fæces nitrogen, but each investigation lasted seven days only. P. Müller (45), Van Noorden and Ritter (64), Mann (41),

Kornblum (32), and Baginsky (3a), all publish researches extending over periods of a few days. In all the total nitrogen output was determined, but in many the nitrogen in the food was wholly or partly taken from diet tables, and in none was the urea determined. Korkounov (31), Evdokimov (13), Garine (16), and Grigoriev (18), have published researches upon the subject, but their papers, which are in Russian, are not easily accessible, and we have not been able to obtain full particulars of their work.

The case we have investigated was one of subacute parenchymatous nephritis, in a boy aged seven, under the care of Dr. Taylor and Dr. Fawcett, in John ward, Guy's Hospital. The research was continuous for forty-one days, and included a period of mild uræmia. The estimations carried out daily were: (1) The total nitrogen in the urine. (2) The urea. (3) The uric acid. (4) The coagulable proteid. (5) The total nitrogen in the fæces. (6) The nitrogen in the milk supply. From time to time additional estimations were necessary, as other articles of food were added to his diet.

No attempt was made to estimate the nitrogen loss by the skin, because we felt that any method of doing so that we could devise must be too inaccurate to correspond with the accuracy we hoped to attain in our other work. The boy was suffering from psoriasis, which may have made the skin loss more than usual.

We have divided the report of our work into the following sections:—

- I. A clinical account of the case.
- II. The methods we have used.
- III. The detailed tables of our results.
- IV. The urea.
- V. The uric acid.
- VI. The coagulable proteid.
- VII. The "other bodies."
- VIII. The fæces.
- IX. The unrecovered nitrogen.
- X. General conclusions.
- XI. References.

SECTION I.

A Clinical account of the Case.

W. W., male, aged 7 years, was admitted under the care of Dr. Fawcett, January 10th, 1901, for cough, dropsy, and albuminuria. He was the eighth child of a family of twelve, of whom only two besides the patient were living.

There was a history of varicella three years previously, and of attacks of psoriasis. There was no history of diphtheria or scarlet fever. His present illness began about October, 1900, and was attributed by his mother to getting his feet wet. His feet began to swell about December 10th, and his eyelids and face were swollen about December 27th. No history of hæmaturia could be obtained.

On admission (January 10th), his pulse was 112, respirations 40 per minute, and temperature 100·4° F. He had a good deal of bronchitis and general œdema. The cardiac sounds were normal, and the pulse tension was not particularly high.

The urine had a sp. gr. of 1040, and contained about 12 parts per 1000, of albumen (by Esbach). There was a large deposit of urates.

He had numerous patches of psoriasis on the extensor surfaces of his arms and legs, and some on the trunk. He was very drowsy, and during the first two or three days in the ward he slept much, and passed little urine.

Under treatment his condition improved, and on January 17th he was passing much more urine, and had very little œdema. After this his condition varied considerably. The albumen never disappeared from the urine, and on several occasions as large a proportion as 14 parts per 1000 (by Esbach) was recorded. Granular and epithelial casts were found.

The research began on February 19th. For some days previously his diet had been milk only. He had slight œdema

of the feet and legs. The abdomen was large, but there was no ascites. He had no bronchitis. The cardiac sounds were normal. There were numerous patches of psoriasis on the legs, arms and trunk; the face and scalp being free. He had a hot bath every night and was taking the following drugs:—

- | | | | |
|--------|---------------------------|-----|---------|
| (1). R | Ferri et Ammonii Citratis | ... | gr. vi. |
| | Liquoris Arsenicalis | ... | ℥ iv. |
| | Liquoris Ammonii Acetatis | ... | ℥ j. |
| | Potassii Citratis | ... | gr. x. |
| | Syrupi Aurantii | ... | ℥ ss. |
| | Aquam ad | ... | ℥ j. |

The mixture given three times a day.

- (2). R Pulveris Scammonii Compositi (B.P.) gr. x.

The powder given, in a cachet, once a day.

February 19th. Bowels Opened (=B.O.) 1.

" 20th. B.O. 2.

" 21st. B.O. 6.

" 22nd. The mixture (1) was omitted, and the following given for the rest of the time:—

- | | | | |
|--------|---------------------------------|------|---------|
| (3). R | Potassii Citratis | ... | gr. xx. |
| | Aquam Menthæ Piperitæ (B.P.) ad | ℥ j. | |

The mixture given three times a day.

February 23rd. The powder (2) was not given to-day. B.O. 4.

" 24th. B.O. 2.

" 25th. The patient vomited a very small amount of clear watery fluid. B.O. 4.

" 27th. B.O. 5.

" 28th. The patient was drowsy. He slept about one and a half hours during the daytime. The powder (2) was again omitted. B.O. 1.

March 1st. A few patches of psoriasis appeared on the face, and fresh patches on the trunk. B.O. 2.

" 2nd. The powder (2) was reduced by one-half, and from this date he took 5 grains each night, in a cachet, unless otherwise stated. He vomited again to-day, a very small quantity of clear fluid of acid reaction. Those who

saw him agreed that he had become distinctly uræmic; it is not easy to bring this out clearly in a short report, as the change was gradual. But the amount of urine passed fell markedly, and he took food very badly.

- March 3rd. He was very drowsy and slept about three hours in the daytime. He complained of headache, and of feeling sick. He took food badly. B.O. 1.
- " 4th. He vomited three times, all the vomits being quite small. There was slight œdema of the face. He slept about one and a half hours during the morning, and took little food all day. B.O. 5.
- " 5th. He vomited once. Some œdema of face.
- " 6th. Œdema of face less. Patient was brighter. He vomited once, but took his food a little better. B.O. 2.
- " 7th. Patient complained of headache; his psoriasis was getting worse. The hot baths were discontinued from this date. He vomited once. B.O. 2.
- " 8th. The œdema of legs and feet was getting worse. There was also slight œdema of the face. B.O. 1.
- " 9th. Patient complained of headache and feeling sick. B.O. 6.
- " 10th. Patient vomited once, and took his food very badly. There was a great deal of swelling on both sides of the face, over the parotid glands.
- " 11th. The parotid swelling was less marked, and he took food better. The powder was not given. B.O. 4.
- " 12th. Much general œdema, and abdomen larger. He complained of headache; vomited slightly twice; and during the night passed urine into the bed in his sleep. He took little food. B.O. 2.

March	13th.	Œdema less. He vomited once
"	14th.	Fair amount of general œdema. Face less œdematous; abdomen very large. He complained of slight headache. He began taking his food better; and from his general condition it was agreed that the uræmic symptoms had markedly abated
"	15th.	Headache. Œdema of face slight; but marked in feet and legs. He took his food well. B.O. 3.
"	16th.	Headache, and feeling of sickness. Marked œdema of legs and feet. A fine red papular rash appeared on the shoulders, and caused irritation and itching.
"	17th.	Rash more marked. Vomited once. B.O. 4.
"	18th.	Patient was brighter and more cheerful, complained of irritation from the rash; was taking his food well.
"	19th.	Slept for one hour during morning. Some œdema of face. B.O. 2.
"	20th.	Rash still present, but it caused less irritation. A good deal of œdema of the face, loins, legs and feet. B.O. 1.
"	21st.	Œdema about the same. Psoriasis getting better and rash disappearing. B.O. 4.
"	22nd.	Œdema about the same. B.O. 4.
"	23rd.	Œdema slightly less; both the rash and the psoriasis were disappearing. B.O. 5. Patient did not sleep well during the night, but took his food very well.
"	24th.	Œdema of feet, legs, and loins persisted; abdomen very large, but no ascites found. The powder was not given. B.O. 3.
"	25th.	Patient vomited slightly once, but continued to take food well. B.O. 3.
"	26th.	Patient was drowsy this morning. There was some œdema of the face and eyelids. The powders were discontinued from to-day. B.O. 1.

March	27th.	Œdema less. Patient brighter. B.O. 1.
"	28th.	Only slight œdema of the feet and legs. Psoriasis almost gone. Rash quite gone. B.O. 1.
"	29th.	B.O. 1.
"	30th.	Some headache in the afternoon. Patient was not quite so well and took less food. B.O. 2.
"	31st.	Patient complained of headache, was drowsy and less well. He took less food, and seemed to be entering upon a second attack of uræmia.
April	1st.	End of period of observation. As nearly as could be judged there was about the same amount of œdema of the feet and legs as at first. Œdema of legs and feet was present all through, but has not been noted every day. Although the abdomen became very large at times, there was never any definite ascites, nor were there any signs of fluid in the chest.

The vomits recorded have always been very small, on no occasion more than about 30 c.c., and generally much less.

The temperature was taken per rectum every four hours throughout, at the same times the pulse and respirations were counted. The highest temperature recorded was 99·4° This was reached on seven occasions, namely :—

March	24th.	2 p.m.	Pulse 100	Respiration 30
"	25th.	6 p.m.	104	30
"	28th.	6 p.m.	96	34
"	29th.	6 p.m.	104	30
"	30th.	6 p.m.	96	28
"	31st.	6 p.m.	100	28

The lowest temperature recorded was 96°, on March 16th, at 2 a.m., when pulse = 66, respiration = 15.

Except where otherwise stated, the patient slept well throughout the night (ten to eleven hours). He took no exercise during the period he was under observation, and except on a few occasions when he was allowed to sit up for an hour or two, wrapped

in a blanket, he was kept in bed the whole time. He never had any hæmaturia.

SECTION II.

The methods we used.

*by one of us.

I. *Supervision of the patient.*

The patient was under the charge of two special nurses, of whom one or other was always in attendance, day and night. Each period of twenty-four hours began at 9 a.m.

II. *Administration and measurement of the food.*

(a). *Milk*.—This formed the major part of the diet throughout. The day's supply, 2000 c.c. was measured out* at 9 a.m., with a litre flask, and stored in a clean dry Winchester quart stoppered bottle. A sample of the same milk was taken for analysis each day. The milk required by the patient from time to time was taken, by the nurse, from the quantity measured out; any portion unconsumed being at once returned to the bottle. A special drinking vessel was used, which was washed only at the end of each day. The quantity remaining in the Winchester quart at 9 a.m. of the next day was drained* into a graduated cylinder, and the amount read off. This, deducted from the amount measured out, gave the milk consumed.

The following articles were subsequently added to his diet:—

(b). *Bread*.—A quantity, free from crust, was placed in a closely covered tin, and the tin and bread weighed together.* The bread eaten by the patient was taken by the nurse from this tin, and the remainder, with the tin, was weighed* at the end of each day. The difference between the two weighings gave the amount consumed by the patient. A sample of the same bread was taken for analysis.

(c). *Biscuit*.—The dinner biscuits were small and dry, and were weighed in the same manner as the bread. A sample was analysed.

The Garibaldi biscuits were found to vary extremely little in weight. The following are the weights :—

(a). Of sixteen biscuits together ... = 327·70 grms.

From which the average of each biscuit = 20·48 grms.

(β). Of single biscuits, taken at random :—

(i) = 20·50 grms.

(ii) = 20·30 grms.

(iii) = 20·40 grms.

(iv) = 20·53 grms.

The number of these biscuits consumed each day was counted; and this, multiplied by the average weight of a single biscuit, gave the quantity consumed. A sample was taken for analysis.

(d). *Liebig's extract of meat*.—A sample having been taken for analysis, the remainder was weighed in the containing jar. It was kept closely covered; a spoonful or more was taken by the nurse as required; and, to ensure that it all reached the patient, it was stirred into boiling water with the same spoon, until dissolved. The jar with its contents was weighed* each day, the successive differences giving the daily consumption.

(e). *Other articles*.—The drugs given to the patient are mentioned in the clinical account. The amount of nitrogen they contained was calculated to be so small, that we thought it might be neglected.

Water, both plain and aerated, was supplied to the patient when he asked for it; the quantity was only roughly measured, by the nurse, in fluid ounces.

He was also allowed a few sweets (acid drops) during a portion of the time. The nitrogen in these was practically nil.

III. *The collection and measurement of the urine, fæces and vomits.*

(a). *The urine*.—Previous to the beginning of the research, the patient was trained by his nurses not to defæcate without having previously passed all his urine. Fæces and urine were thus collected quite separately. A special vessel, washed only at the end of each day, was kept for his use; after each micturition the nurse poured the urine into a

clean stoppered Winchester quart bottle, reserved for that day's urine. At 9 a.m., the end of each day, the bottle containing the whole quantity collected during the previous twenty-four hours, was handed over to us.

The amount was measured* in a graduated cylinder, to the nearest c.c., and a sufficient quantity of the mixed urine taken for the different analyses.

The reaction to litmus paper was recorded,* and the specific gravity read off on a urinometer. For the last thirty-one days the specific gravity was also ascertained by weighing a measured volume (*vide* albumen estimation).

(b). *The fæces*.—Each motion was passed into a separate porringer, and as many of these as had been used were handed over to us at the end of each twenty-four hours. The contents of the whole number were washed with a jet of distilled water into a large weighed evaporating basin, and well mixed with a glass rod, hydrochloric acid being added until they distinctly reddened litmus paper. They were then put into a hot water oven and kept at about 80° C., being stirred from time to time until they appeared dry; this took from twelve to thirty-six hours, after which they were further dried for about six hours in a hot air oven, between 100° C. and 110° C. They were allowed to cool in a desiccator over strong sulphuric acid, and then weighed in the evaporating basin, to the nearest decigram.

The dried fæces were powdered as finely as possible in a mortar, and a sample kept for analysis.

(c). *The vomits*.—These were collected separately, in porringers. All were extremely small. Analysed separately at first, they were found to contain so little nitrogen that subsequent vomits were mixed with the fæces, and estimated with them. Further mention of this is made in Section VIII.

IV. *The methods of analysis.*

The pipettes and burettes used were carefully standardised before the experimental work began. Several blank Kjeldahl estimations were made, to determine the correction for the apparatus.

The Kjeldahl method, as described by Kossel (33), was employed for the estimation of the total nitrogen in the milk; bread; Garibaldi biscuit; dinner biscuit; Liebig; urine; and fæces. Two estimations of the milk, urine and fæces were made each day. The results of the analyses of the food stuffs are given in Table II.

The urea was estimated by the Mörner-Sjöquist (44) process, in two 5 c.c. samples each day.

The uric acid was determined by Hopkins' (26) potassium permanganate method, in two 100 c.c. samples each day.

The albumen was estimated by the trichloroacetic acid method from February 20th to 28th inclusive; for the remainder of the time by a heat coagulation process. The following are detailed descriptions of each:—

(a). *The trichloroacetic acid method.*—To two 50 c.c. samples of urine each day, an equal quantity of 10 per cent. trichloroacetic acid was added. After standing, the precipitate was filtered off, and washed with a jet of hot 5 per cent. trichloroacetic acid, until the washings gave no reaction with sodium hypobromite. The filter paper with the precipitate was dried; first on a water bath below 60° C., until apparently dry; then in a hot air oven at 90° C. to 120° C. for four hours; and finally in a desiccator over strong sulphuric acid for four days. It was then weighed, and the known weight of the dry filter paper deducted. The percentage of nitrogen in four samples of the precipitated albumen was determined by Kjeldahl; and from this the nitrogen as albumen in each day's urine calculated.

(b). *The heat coagulation process.*—50 c.c. of urine were run into an india-rubber corked flask, the weight of the cork and flask being known. The urine, cork, and flask were weighed; the difference gave the weight of urine taken, and also its accurate specific gravity. The urine was then boiled, and, while boiling, 5 per cent. acetic acid was added from a burette until no further coagulation occurred. About 5 c.c. was usually added in this way. The flask was corked, allowed to cool, and again weighed.

The coagulated proteid was then removed by filtering, and the filtrate tested with nitric acid to be sure that none remained. The nitrogen in two 10 c.c. samples of the proteid free urine was estimated by Kjeldahl.

But 10 c.c. of this boiled urine = 10 c.c. of the original urine $\times \frac{\text{weight after boiling.}}{\text{weight before boiling.}}$ Therefore the nitrogen found in 10 c.c. of boiled urine was multiplied by the factor: $\frac{\text{weight before boiling}}{\text{weight after boiling}}$, to give the nitrogen in 10 c.c. of original urine freed from coagulable proteid. The total nitrogen in the urine being known, the difference between this and the nitrogen in the proteid free urine gave the coagulable proteid nitrogen.

The greatest correction was—

$$\frac{50.462 \text{ (weight in grms. before boiling)}}{56.043 \text{ (weight in grms. after boiling)}};$$

usually it was much less.

SECTION III.

Tables of results.

Tables I. and II. give in extenso our experimental results and calculations from them. They are referred to in the different sections, and summaries are there made. The meaning of the figures is explained by the headings of the columns.

The following are the notes we wish to make about these two tables.

1. When not otherwise specified, two estimations were made in every case, and the mean taken. Whenever, in the following notes, it is stated that the higher of the two values was taken, this was because the difference between the two observations was more than 1 per cent. from the mean.

2. *The volume of urine.*—The figures are for urine actually measured, except on :—

March 9th, when 285 c.c. was measured and nurse reported zi. (=28 c.c.) spilled.

March 12th, when 267 c.c. was measured, and nurse reported ziii. (=85 c.c.) passed into bed.

ABL

THE NITROGEN		THE URIC ACID RELATION.		THE UREA NITROGEN RELATIONS.	
in nd .	Nitrogen in Fæces.	The Ratio. Uric Acid Nitrogen : to Total Nitrogen in Urine.	The Ratio. Uric Acid (as such) : to Urea (as such).	The total Nitrogen in the Urine being regarded as 100, the Urea Nitrogen equals :—	The total Nitrogen in the Urine, minus the Coagulable Proteid Nitrogen being regarded as 100, the Urea Nitrogen equals :—



March 25th, when 1490 c.c. was measured, and nurse reported 3ii. (=7 c.c.) spilled.

March 26th, nurse reported a small quantity spilled, for which we made no allowance.

3. *The specific gravity of the urine.*—When no decimal place is given, there was a urinometer reading only. Otherwise the figure is calculated from the weight of a measured volume.

4. *The milk.*—March 3rd, nurse reported a small quantity spilled. We did not correct for this, so that it was calculated as having been taken by the patient.

One estimation only: February 19th, March 3rd, March 21st.

The higher value taken: March 19th.

5. *The vomits.*—All were extremely small. They were lost on February 25th (1), February 28th (1), March 3rd (3). They were separately estimated, and the nitrogen added to the fæces on the following days:—

March 5th = 0.0483 grms. nitrogen.

March 6th = 0.0406 "

March 14th = 0.0629 "

They were estimated along with the fæces on March 10th, 12th, 17th and 24th.

6. *The fæces.*—On March 3rd the amount was so small, one Kjeldahl only was made. There were three estimations February 20th, 22nd and 23rd.

7. *Total nitrogen in urine.*—

One estimation only on March 4th and 7th.

Higher value taken on March 3rd and 21st.

8. *Urea nitrogen.*—

One estimation only on February 25th, 26th and 28th;

March 6th, 7th, 12th, 22nd and 29th.

Higher value taken on March 2nd, 5th, 11th and 28th.

9. *Uric acid nitrogen.*—

One estimation only on March 4th.

Higher value taken on February 21st, March 1st, 2nd, 12th, 19th and 22nd.

10. *Proteid free urine.*—

No estimation at all on February 19th and March 6th.

Higher value taken on March 9th, 10th, 21st and 25th.

The columns of the tables are arranged so as to correspond ; the urine and fæces figures, for example, are for the day on which the patient passed the urine and fæces ; the milk figures for the day on which he consumed the milk.

The total intake and the total output nitrogen for each day, the progressive nitrogen deficit, and the patient's weight for each day, are represented graphically in the chart.

SECTION IV.

The Urea.

In Text-Books of Medicine (11) the statement is often met with, that in Bright's disease, and particularly in uræmia, the urea is diminished. Authors do not specify whether this diminution is relative as well as absolute.

That the absolute quantity of urea excreted by a person with uræmia should be less than that by a healthy individual, is to be expected, because his food nitrogen is less. But he may be excreting quite as large a proportion of urea as would a normal person upon the same diet.

We have found no experiment in which a healthy boy, of the same age as our patient, took exactly the same amount of food nitrogen as he did. Therefore we cannot say whether a healthy boy, and a boy with subacute Bright's disease, each having the same quantity of food nitrogen, would excrete equal amounts of urea, or not. But we can determine the percentage of the total nitrogen in the urine which is in the form of urea ; and thus find out whether the proportion of urea is diminished in Bright's disease and uræmia, or not.

In the following table are summarised the results obtained by different observers, in healthy persons. Few complete investigations by the Mörner-Sjöquist process have yet been carried out ; we have, therefore, included some less recent figures, obtained by modified hypobromite methods. In one case only was the diet solely milk. In all cases, the total nitrogen was determined by Kjeldahl :—

TABLE III.—The Urea Nitrogen Relations found by different Observers in the Urine of Healthy Persons.

Observer.	Date.	Patient's		Diet.	Duration of Observation in days.	Method of Estimating Urea.	The urea nitrogen expressed per cent of total nitrogen in urine.		
		Sex.	Age.				Mini- mum.	Maxi- mum.	Aver- age.
Pfäuger and Bohland (46)	1886	Themselves		Mixed ...	?	Pfäuger-Bohland	84.0	90.3	86.8
Horton-Smith (28)	1887	Himself		Peptonised milk	3	Pfäuger-Hüfner	83.8	87.0	85.3
Bohland (6)	1888	Himself		Mixed ...	3	"	81.2	83.8	82.2
Schuitze (55)	1889	Himself		Mixed ...	?	Pfäuger-Bohland	—	—	85.0
Gumlich (20)	1893	Himself		Various...	?	Schlosing-Bohland...	83.8	89.6	86.8
Camerer (8)	1896	Himself		Mixed ...	24	Phosphotungstic acid	78.8	88.8	84.0
"	"	Himself		Mixed ...	14 observations	Hüfner	83.8	90.5	87.3
"	"	Himself		Meat ...	5	"	—	—	93.8
"	"	Himself		Vegetable	2	"	—	—	86.9
"	"	Himself		Vegetable	2	"	—	—	85.8
"	"	Himself		Meat ...	3	"	—	—	88.3
Dunlop (12)	1896	M 85		Mixed ...	4	Pfäuger-Bohland	81.0	88.9	86.2
"	"	M 42		Mixed ...	3	"	77.5	84.1	80.1
"	"	M 40		Mixed ...	3	"	77.5	84.3	81.2
Camerer (9)	1897	M 56		Mixed ...	?	Mörner-Sjöquist	—	—	89.9
"	"	F 16		Mixed ...	?	"	—	—	86.3
"	"	M 63		Mixed ...	?	"	—	—	88.1
"	"	Himself		Mixed ...	7 observations	"	87.6	93.7	90.3
Goodbody (19)	1900	M ?		Mixed ...	6	Mörner-Sjöquist	70.5	89.3	84.0
"	"	M ?		Mixed ...	4	"	84.2	92.0	87.6
Macleod (39)	1901	?		Flesh ...	?	Mörner-Sjöquist	—	—	96.1
"	"	?		Creatinin free ...	?	"	—	—	82.5
Joslin (39)	"	F ?		Mixed ...	15	Squibbs	80.6	94.4	88.9
Hale White and Spriggs (22)...	"	F 38		Mixed ...	55	Mörner-Sjöquist	67.5	96.4	87.3

Referring to the results obtained by the Mörner-Sjöquist process only, it will be seen that the percentage of the total nitrogen in the urine which is present as urea nitrogen, may, in health, be as low as 67·5 per cent., or as high as 96·4 per cent. These are extreme variations. Hale White and Spriggs, in a series of twenty-seven consecutive observations upon the same person, found an average of 87·3 per cent.

The averages obtained by the other workers quoted, vary from 80·1 per cent. to 93·8 per cent.

In none of the metabolism experiments upon Bright's disease, have we found the urea estimated at the same time as the total nitrogen. Prior (50) gives figures for the urea, by Pflüger's method, in some cases; but, when reduced to nitrogen, the urea nitrogen and the total nitrogen are identical. P. Müller (45), we believe, estimated the urea; but we have been unable to obtain the inaugural dissertation which contains his researches.

Gumlich (20), in occasional samples of urine from patients with chronic parenchymatous nephritis, estimated the urea as nitrogen not precipitated by phosphotungstic acid; and found the proportion of urea nitrogen to vary from 72·3 per cent. to 88·9 per cent. of the total nitrogen in the urine. He made no allowance for the coagulable proteid nitrogen.

Our own results are given in detail in Table I. We have calculated the percentage relation of the urea nitrogen both to the total nitrogen in the urine, and to the urinary nitrogen remaining after deduction of that present as coagulable proteid. Below we give the results summarised:—

- (1). For the whole period.
- (2). For the period of uræmia.
- (3). For the non-uræmic period.
- (4). For the different diets.

TABLE IV.

	Per cent. of urea nitrogen referred to total urinary nitrogen.	Per cent. of urea nitrogen referred to total urinary Nitrogen minus congealable proteid nitrogen.		Urea as such, total in grammes.		Grammes of urea as such in 100 c.c. urine.	
	DATE.	DATE.	DATE.	DATE.	DATE.	DATE.	DATE.
Whole period.—February 19th to March 31st.	Maximum	Feb. 24	Feb. 24	Feb. 21	14.02	Mar. 4	2.77
	Minimum	Feb. 20	Mar. 21	Mar. 4	5.76	Mar. 18	0.90
	Average	11.54	...	1.27
Period of Mild Uremia.—March 2nd to March 13th	Maximum	Mar. 5	Mar. 5	Mar. 2	13.36	Mar. 5	3.13
	Minimum	Mar. 11	Mar. 11	Mar. 4	5.76	Mar. 13	1.23
	Average	8.32	...	2.07
Whole period minus uremic period.—February 19th to March 1st, and March 14th to March 31st	Maximum	Feb. 24	Feb. 24	Mar. 27	18.00	Feb. 25	2.36
	Minimum	Feb. 20	Mar. 21	Mar. 16	8.18	Mar. 28	0.55
	Average	12.58	..	1.01
Milk diet only.—February 19th to March 4th, and March 18th to March 22nd	Maximum	Feb. 24	Feb. 24	Feb. 21	14.02	Mar. 4	2.77
	Minimum	Feb. 20	Mar. 21	Mar. 4	5.76	Mar. 18	0.90
	Average	11.54	..	1.27
Diet of biscuits or bread and milk.—March 5th to March 17th.	Maximum	Mar. 14	Mar. 15	Mar. 17	11.72	Mar. 5	3.13
	Minimum	Mar. 11	Mar. 11	Mar. 12	6.26	Mar. 16	1.09
	Average	8.57	...	1.67
Diet of milk, biscuits and Liebig.—March 23rd to March 31st	Maximum	Mar. 29	Mar. 27	Mar. 27	18.00	Mar. 23	1.18
	Minimum	Mar. 28	Mar. 28	Mar. 31	10.39	Mar. 28	0.55
	Average	14.88	...	0.79

At first sight, the percentage of urea nitrogen appears distinctly lower than in health; the average for the whole period being 75·6 per cent., as against 87·3 per cent. in Hale White and Spriggs' case.

But, when we deduct from the total nitrogen of the urine, that of the abnormal constituent, coagulable proteid, the urea nitrogen forms 83·4 per cent. of the remainder; the extreme daily variations being 71·8 per cent. and 94·6 per cent. Regarding the coagulable proteid, in this way, as an accidental impurity, the proportion of urea nitrogen, in our case, was, therefore, within the limits found in health.

As regards the uræmic period, the urea nitrogen formed, on an average, 82·03 per cent. of the urinary nitrogen after deduction of that in the coagulable proteid. This is slightly less than 84·42 per cent., the average proportion for the remainder of the time. But in Section V. it is shown that the proportion of uric acid was at its maximum during the uræmic period; this will partly account for the slightly lower proportion of urea nitrogen. In our patient, therefore, the onset of uræmic symptoms was accompanied by no marked relative decrease in the urea nitrogen.

The urea in 100 c.c. urine, on the other hand, rose considerably at this time. The average, for the uræmic period, was 2·07 grms. of urea in 100 c.c. urine; whereas, for the remainder of the time, it was 1·01 grms. The reason of this was the smaller volume of urine passed, and its consequent greater concentration.

The influence of the different diets is also little marked. It will be seen, in Table IV., that the urea nitrogen formed a slightly greater percentage of the whole when Liebig was given, than without it; but, whether the coagulable proteid nitrogen be reckoned, or be previously deducted, the variations caused by diet were slight.

The conclusions, therefore, which we draw from this section of our work are, that, in our patient with subacute nephritis:—

(1). There was no diminution of the proportion of urea nitrogen to the total nitrogen of the urine, if the coagulable proteid be regarded as an accidental body, and its nitrogen first deducted from the total; but that, reckoning the nitrogen of this abnormal

body in the total, the proportion of urea nitrogen was about 10 per cent. below the normal.

(2). The proportion of urea nitrogen to total urinary nitrogen was not appreciably lower when uræmic symptoms showed themselves, than at other times; the total urea nitrogen was less during uræmia, as the food was less; whilst the percentage of urea in the urine rose considerably.

(3). The addition of bread, biscuit, or Liebig's extract of meat to a purely milk diet caused no corresponding alteration in the proportion of the urea nitrogen.

SECTION V.

The Uric Acid.

In Osler's Principles and Practice of Medicine, p. 409, there is a quotation from Kolisch (30) to the effect that in nephritis the uric acid is diminished, and the xanthin bases in the urine increased. In his original paper Kolisch states that in nephritis the total alloxuric bodies remain constant, but that there is an increase in the xanthin bases (xanthin, hypoxanthin, paraxanthin, adenin, guanin, carnin), at the expense of the uric acid. He quotes Baginsky (3), who found that in children with nephritis the xanthin in the urine increases.

Kolisch and Dostal (30) estimated the total alloxuric bodies by the method of Krüger and Wulff (34), the uric acid by the Ludwig (38) Salkowski (58a) process. In health they give the proportion :

$$\frac{\text{uric acid nitrogen}}{\text{xanthin base nitrogen}} = \frac{4}{1};$$

and this is confirmed by Kossel's pupils, quoted by Zuelzer (69), whilst Weintraud (67) found $\frac{5}{7}$ and $\frac{7}{7}$. In nephritis their analyses showed for single day estimations with different patients:—

$$\frac{\text{Uric acid nitrogen}}{\text{Xanthin base nitrogen}} = \frac{\cdot 24}{1}; \frac{\cdot 24}{1}; \text{ and } \frac{\cdot 59}{1};$$

The nitrogen of the total alloxuric bodies in these cases was 0.152 grms., 0.220 grms., and 0.312 grms. respectively, whilst in health they found 0.260 grms. They draw the conclusion that one of the functions of the kidney is to convert the xanthin bases into uric acid, and that in nephritis this change does not take place.

Frerichs (15), Bartels (5), Dickinson (10), Fleischer (14), Wagner (66), and Rosenstein (51), using Heintz's (23) method, found the uric acid diminished in Bright's disease. Géza Fodor (17), by the Ludwig-Salkowski (53a) process, obtained similar results to those of Kolisch and Dostal as regards xanthin bases and uric acid, and further found that the fall of uric acid in the urine of nephritis was unaccompanied by any rise of uric acid in the blood.

On the other hand, many investigators have been unable to demonstrate any such fall. Vogel (65), though he used Heintz' method, found as much uric acid in acute nephritis as in health; and the following more recent workers, employing the Ludwig-Salkowski or Hopkins (26) processes, confirm this (*See Table V*).

Zuelzer (69) further states that he never found the uric acid in Bright's disease less than the xanthin bases, his ratio—

$$\frac{\text{Uric acid nitrogen}}{\text{Xanthin base nitrogen}} \text{ varying from } \frac{1.3}{1} \text{ to } \frac{35}{1} \text{ to } \frac{\infty}{1};$$

whilst Umber's (62) figures, for healthy persons, show variations between $\frac{3.7}{1}$ and $\frac{36.6}{1}$.

The following table shows the values for uric acid, in health, obtained by modern methods. (*See Table VI*.)

It will be seen that there are very wide variations in the uric acid, even in health. The ratio $\frac{\text{uric acid}}{\text{urea}}$ in Table VI. varies from $\frac{1}{34}$ to $\frac{1}{30}$; and the ratio $\frac{\text{uric acid nitrogen}}{\text{total urinary nitrogen}}$ from $\frac{1}{24}$ to $\frac{1}{20}$.

TABLE V.

Observer.	Date.	Total daily uric acid in grammes.	Form of Nephritis.	Uric acid relations;—		Methods.		
				Uric acid nitrogen to total urinary nitrogen.	Uric acid as such to urea as such.	For total nitrogen.	For uræa.	For uric acid.
Stadthagen (59)	1887	.3 to .5	Nephritis...	—	$\frac{30}{30}$ to $\frac{30}{30}$	—	?	Salkowski
Prior (50)	1891	.6 to .7	Acute nephritis ...	$\frac{38}{103}$ to $\frac{47}{100}$	—	Kjeldahl	—	Fokker Salkowski
"	"	.3 to .5	Chronic interstitial	$\frac{103}{86}$ to $\frac{30}{30}$	—	"	—	"
"	"	.3 to .9	Sclerosis of kidney	$\frac{86}{137}$ to $\frac{30}{78}$	—	"	—	"
"	"	.2 to .5	Chronic parenchymatous	—	$\frac{30}{30}$ to $\frac{47}{30}$	"	—	"
Van Ackeren (68)	1892	.3 to 1.0	Acute nephritis ...	—	$\frac{30}{30}$ to $\frac{47}{30}$	—	Pfütger Liebig	Ludwig Salkowski
"	"	.3 to .7	"	—	$\frac{30}{30}$ to $\frac{47}{30}$	—	"	"
"	"	.7 to 1.0	"	—	$\frac{30}{30}$ to $\frac{47}{30}$	—	"	"
"	"	.3 to 1.2	"	—	$\frac{30}{30}$ to $\frac{47}{30}$	—	"	"
"	"	.2 to .5	Chronic parenchymatous	—	$\frac{30}{30}$ to $\frac{47}{30}$	—	"	"
"	"	.6 to .9	"	—	$\frac{30}{30}$ to $\frac{47}{30}$	—	"	"
"	"	.4 to .6	"	—	$\frac{30}{30}$ to $\frac{47}{30}$	—	"	"
"	"	.7 to 1.5	Granular kidney	—	$\frac{30}{30}$ to $\frac{47}{30}$	—	"	"
"	"	.6 to .8	"	—	$\frac{30}{30}$ to $\frac{47}{30}$	—	"	"
"	"	.2 to .5	"	—	$\frac{30}{30}$ to $\frac{47}{30}$	—	"	"
Zuelzer (69)	1896	.5	?	$\frac{47}{30}$	$\frac{30}{30}$ to $\frac{47}{30}$	Kjeldahl	—	Ludwig Salkowski
"	"	.5	?	$\frac{47}{30}$	—	"	—	"
"	"	.7	?	$\frac{47}{30}$	—	"	—	"
"	"	.4	?	$\frac{47}{30}$	—	"	—	"
Milroy and Malcolm (43)	1898	.1 to .6	Granular kidney	$\frac{47}{30}$	—	Kjeldahl	—	Ludwig Salkowski
Haig (21)	1900	3.7 grains	Nephritis (in a child of 5)	—	$\frac{30}{30}$	—	Hüfner ...	Haycraft Salkowski

TABLE VI.—*Uric Acid Relations in Healthy People.*

Observer.	Date.	Total daily uric acid in grammes.	Diet.	Uric acid relations.		Methods.		
				Uric acid nitrogen to total nitrogen in urine.	Uric acid as such to urea as anch.	For total nitrogen.	For urea.	For uric acid.
Horton-Smith (28)	1888	.3 to .4	Milk	—	$\frac{8}{15}$	—	Pfäuger Hüfner	Salkowski
"	"	.3 to .4	Peptonised milk	$\frac{13}{15}$	$\frac{17}{15}$	Kjeldahl	"	"
Yvon and Bertloz (68)	1888	.6 to .8	Mixed	$\frac{17}{15}$	$\frac{30}{15}$	"	"	"
Schultze (55)	1889	.7 to 1.5	"	—	$\frac{17}{15}$ to $\frac{16}{15}$	—	"	"
Pott (47)	1889	.5 to 1.2	"	$\frac{15}{15}$ to $\frac{16}{15}$	$\frac{17}{15}$ to $\frac{16}{15}$	Kjeldahl	Schlösing Bohland	Fokker Salkowski
Schöndorff (54)	1890	.7 to 1.3	"	$\frac{17}{15}$ to $\frac{16}{15}$	$\frac{17}{15}$ to $\frac{16}{15}$	Kjeldahl	—	Fokker Salkowski
Herringham & Groves (24)	1891	—	"	—	$\frac{17}{15}$	—	Hypobromite, corrected	Ludwig Salkowski
Umber (62)	1896	.5 to 1.6	"	$\frac{17}{15}$ to $\frac{16}{15}$	—	Kjeldahl	—	Ludwig Salkowski
"	"	.7 to 1.0	"	$\frac{15}{15}$ to $\frac{17}{15}$	—	"	—	"
"	"	.4 to .9	"	$\frac{15}{15}$ to $\frac{17}{15}$	—	"	—	"
Strauss (60)	1896	.4 to .7	"	$\frac{15}{15}$ to $\frac{17}{15}$	—	Kjeldahl	—	Salkowski
"	"	.9 to 1.4	Mixed and Liebig	$\frac{15}{15}$ to $\frac{17}{15}$	—	"	—	"
Hopkins and Hope (27)	1898	.5 to .8	Various	$\frac{15}{15}$ —	$\frac{17}{15}$ to $\frac{16}{15}$	—	Knop-Hüfner & Möner Sjöquist	Hopkins
Smith-Jerome (57)	1898	.4 to .8	Mixed	$\frac{15}{15}$ to $\frac{16}{15}$	—	Kjeldahl	Möner Sjöquist	Ludwig Salkowski
Bain and Edgcombe (4)	1898	.7	"	$\frac{15}{15}$ —	$\frac{17}{15}$	—	Hypobromite	Hopkins
Milroy and Malcolm (43)	1898	.5	"	$\frac{15}{15}$ —	$\frac{17}{15}$	Kjeldahl	"	Ludwig Salkowski
Goodbody (19)	1900	.6 to .8	"	$\frac{15}{15}$ to $\frac{16}{15}$	$\frac{17}{15}$ to $\frac{16}{15}$	Kjeldahl	Möner Sjöquist	Hopkins & Salkowski
Haig (21)	1900	.7 to .8	"	$\frac{15}{15}$ to $\frac{16}{15}$	$\frac{17}{15}$ to $\frac{16}{15}$	Kjeldahl	Hüfner ...	Haycraft Salkowski
Smith-Jerome (58)	1900	.5	Mixed	$\frac{17}{15}$	—	—	—	Ludwig Salkowski
Tunnicliffe and Rosenheim (61a)	1900	.6	Mixed and Liebig	$\frac{17}{15}$ to $\frac{16}{15}$	—	—	—	Ludwig Salkowski
"	"	.05 to .25	Milk chiefly	$\frac{17}{15}$ to $\frac{16}{15}$	—	—	—	"
"	"	.04 to .22	"	$\frac{17}{15}$ to $\frac{16}{15}$	—	—	—	"
"	"	.18 to .23	"	$\frac{17}{15}$ to $\frac{16}{15}$	—	—	—	"
"	"	.05 to .19	"	$\frac{17}{15}$ to $\frac{16}{15}$	—	—	—	"

$\frac{1}{38}$. In Table V., of Bright's disease, the ratio $\frac{\text{uric acid}}{\text{urea}}$ ranges from $\frac{1}{34}$ to $\frac{1}{7}$; and $\frac{\text{uric acid nitrogen}}{\text{total urinary nitrogen}}$ from $\frac{1}{137}$ to $\frac{1}{39}$. Therefore Kolisch's statement, quoted by Osler, is not supported by the results of more recent analyses.

Our own results are given in full in Table I. The most remarkable point, perhaps, is the almost constant, and relatively high ratio $\frac{\text{uric acid}}{\text{urea}}$ during the period of mild uræmia, March 2nd to 13th inclusive. This ratio is quite as high, but slightly less constant, during the last nine days of the experiment, when Liebig's meat extract was added to the diet. During the remaining twenty days the ratio was very variable, and the mean much lower, as shown in the following summary:—

TABLE VII.

	Ratio of uric acid nitrogen to total nitrogen of urine.			Ratio of uric acid (as such) to urea (as such).			Total daily uric acid, in grams.		
	Max.	Min.	Aver.	Max.	Min.	Aver.	Max.	Min.	Aver.
For the whole experiment 41 days.	$\frac{1}{35}$	$\frac{1}{280}$	$\frac{1}{38}$	$\frac{3}{1}$	$\frac{1}{134}$	$\frac{1}{48}$	·48	·12	·24
For the uræmic period 12 days.	$\frac{1}{38}$	$\frac{1}{90}$	$\frac{1}{52}$	$\frac{3}{5}$	$\frac{1}{45}$	$\frac{1}{38}$	·29	·15	·22
For the remaining period without Liebig 20 days.	$\frac{1}{31}$	$\frac{1}{280}$	$\frac{1}{111}$	$\frac{3}{8}$	$\frac{1}{134}$	$\frac{1}{38}$	·27	·12	·18
For the remaining period with Liebig 9 days.	$\frac{1}{59}$	$\frac{1}{31}$	$\frac{1}{38}$	$\frac{3}{1}$	$\frac{1}{48}$	$\frac{1}{38}$	·48	·31	·39

The ratio $\frac{\text{uric acid nitrogen}}{\text{total urinary nitrogen}}$ is more variable, and of less value, than that of $\frac{\text{uric acid}}{\text{urea}}$, on account of the varying quantities of coagulable proteid in the urine.

The high ratio $\frac{\text{uric acid nitrogen}}{\text{total urinary nitrogen}}$ when Liebig was given, corresponds to the results obtained by Strauss (60) and Smith-Jerome (57) with meat extract feeding in the healthy subject.

We conclude that, in our patient suffering from subacute nephritis:—

(1). The uric acid was not diminished out of proportion to the other urinary constituents.

(2). The variations in the ratio $\frac{\text{uric acid}}{\text{urea}}$ are not greater than those which occur in health.

(3). The ratio $\frac{\text{uric acid}}{\text{urea}}$ became higher, and much more constant, with advent of uræmic symptoms.

(4). The addition of extract of meat to the diet produced, as in health, both an actual and a relative increase in the uric acid.

SECTION VI.

The Coagulable Proteid.

Albumen is present in the urine of Bright's disease in such variable quantities that we have not much to say about it in the present instance. Great care was taken in the estimation of the coagulable proteid nitrogen, but the deductions we can draw are few.

The results have enabled us, however, in Section IV., to deduct albumen nitrogen from total nitrogen, and to find the relation of the urea nitrogen to the remainder, and we concluded that the

coagulable proteid might be regarded as a foreign or adventitious substance.

We made no attempt to differentiate the varieties of coagulable proteid present.

Van Noorden and Ritter (64), estimating the albumen by Scherer's method, conclude from figures tabulated on p. 223 of their paper, that the *kind* of diet has little effect upon the amount of albumen in the urine; but that when there is a *change* of diet there is often an increase in the albumen for a few days after the change. The following table is a summary of our results:—

TABLE VIII.

	Nitrogen as coagulable proteid in 24 hours in grms.	Per cent. of Nitrogen as coagulable proteid referred to total nit- rogen in urine.
Average of whole period (41 days)	0.6536	9.348 per cent.
Average of 11 days in uræmic period (There was no estimation on March 6th)	0.6085	11.640 per cent.
Influence of <i>kind</i> of diet:—		
Average of 18 days milk only	0.7080	9.884 per cent.
Average of 12 days milk and bread or biscuits	0.5700	10.680 per cent.
Average of 9 days milk, biscuit, Liebig	0.6375	7.184 per cent.
Influence of <i>change</i> of diet:—		
Average of last 2 days of milk, before bread	0.5500	—
Average of first 2 days of bread	0.4479	—
Average of last 2 days of milk, before Liebig	0.5417	—
Average of first 2 days of Liebig	0.6948	—

If any conclusions can be drawn at all, we should infer from Table VIII. that the average daily output of coagulable proteid in the urine was almost constant in the different periods; that the addition of bread or Liebig to our patient's diet did not cause it to alter much; that in the uræmic period the average total for each twenty-four hours was approximately the same as the average for the whole period; and that the average daily total was more constant than the average percentage relationship to the total nitrogen in the urine.

It is true that when Liebig was added to the diet there was an increase in the coagulable proteid for the first few days; the last two days, with milk diet before Liebig was given, gave an average of 0.5417 grms. nitrogen as albumen; the average of the first two days of Liebig was 0.6948 grms. nitrogen. The average for the whole Liebig period (0.6375 grms.) on the other hand, was less than the average for the whole period of milk diet (0.7080 grms.) The change, however, from milk to milk and bread or biscuit gave a fall in coagulable proteid nitrogen from 0.5500 grms. to 0.4479 grms. We do not find, therefore, as Van Noorden (64) did, that *change* of diet always caused an increase in the albumen.

The detailed figures in Table I. show the coagulable proteid nitrogen to have been highest on March 1st and 2nd, namely, on the first day of distinct uræmia, and on the day before it, but this may be mere coincidence. It is of interest to compare the figures with the clinical account; it will be seen that the cedema was, as far as we could judge, most marked about March 20th, the coagulable proteid nitrogen was by no means at its maximum at this time. The total amount of coagulable proteid in the urine gave no close index of the cedema present.

SECTION VII.

The nitrogen as "other bodies."

No attempt was made to determine what the "other bodies" were. They were calculated by subtracting the urea nitrogen, the uric acid nitrogen, and the coagulable proteid nitrogen, on the one hand, from the total nitrogen in the urine on the other.

The following table gives the average percentage relation of "other bodies" nitrogen to total urinary nitrogen for the different periods :—

TABLE IX.

	Per cent. of "other bodies" nitrogen referred to total urinary nitrogen as 100.
Whole period :— February 19th to March 31st	13·88 per cent.
Uræmic period :— March 2nd to March 13th	14·52 per cent.
Non-uræmic period :— February 19th to March 1st and March 14th to March 31st	13·60 per cent.
Milk diet only :— February 19th to March 4th and March 18th to March 22nd	14·08 per cent.
Milk and bread or biscuit :— March 5th to March 17th	13·92 per cent.
Milk and biscuit and Liebig :— March 23rd to March 31st	13·24 per cent.

We draw no conclusions from Table IX. The figures are averages ; the daily variations, which were considerable, are given in Table II. It is of interest to note how the average proportion of "other bodies" nitrogen remains almost constant throughout, in spite of alterations in the patient's general condition, and in his diet.

SECTION VIII.

The fæces.

Part of the treatment of Bright's disease is usually directed towards increasing the intestinal elimination, with a view to diminishing the work of the kidneys. Unfortunately it is not yet possible to determine how much of the nitrogen in the fæces is derived from food unabsorbed, and how much, if any, from metabolic end products excreted into the bowel. Nevertheless it is interesting to compare the nitrogen in the fæces of persons suffering from Bright's disease with that in the fæces of healthy people.

Some observers compare the fæces nitrogen with the total nitrogen intake, regarding the former as loss, the difference between the two as the true intake, and the relation between the two as an index of the assimilative power.

This can only be true if no nitrogenous bodies are excreted by the bowel. Pregl (49) has shown that excretory products, such as urea and ammonia, may be found in the intestinal secretion; it seems, therefore, justifiable to compare the fæces nitrogen with the total output of nitrogen in the urine and fæces together, as well as with the nitrogen in the food intake; an additional argument for so doing arises from the fact that in many cases the food nitrogen has been calculated from diet tables, and therefore may be less accurate than the figures obtained by direct Kjeldahl estimations of the urine and fæces. In the following tables both relations are given.

In the first are the results obtained by different observers in normal people. A very large number of fæces determinations has been found in the literature; but for purposes of comparison with our own, only those in which the diet has been chiefly milk have been tabulated; and no case given in which the observation extended over less than three days.

TABLE X.

Name of Observer.	Date.	Patients'			Duration of experiment in days.	Diet.	Nitrogen in faeces.		Average Nitrogen in Faeces per diem in grms.
		Sex.	Age.	Weight in Kgs.			Per cent. referred to Nitrogen Intake.	Per cent. referred to total Nitrogen in Urine and Faeces.	
Rubner (52)	1879	M	27	—	3	Milk only	6.5	6.9	1.0
Hofman (35)	1880	M	—	66	2 x 3*	"	11.1	7.7	1.4
Siatkowski (56)	"	M	—	—	3 x 3*	"	5.8	6.4	1.6
"	"	M	—	—	3 x 3*	"	4.2	4.2	0.8
"	"	F	—	—	3	"	4.9	7.5	1.0
Laptschinsky (35)	"	M	26	55	6	"	4.0	6.4	0.8
"	"	M	46	62	7	"	7.6	6.2	1.3
"	"	M	22	65	6	"	5.1	5.1	1.0
"	"	M	23	67	6	"	11.1	11.6	2.2
"	"	M	24	73	5	"	6.1	5.4	0.8
"	1882	F	12	—	3	"	4.9	4.9	0.5
Camerer (7)	"	F	10	—	3	Milk only	3.7	4.0	0.4
"	"	M	6.3	—	3	"	5.4	4.4	0.5
"	"	F	5.13	—	3	"	7.4	6.5	0.6
"	"	F	4.2	—	3	"	9.2	8.8	0.5
"	1885	M	—	70	3	"	5.5	4.5	0.8
Rudenko (53)	"	M	—	105	5	"	6.7	4.8	0.8
"	"	M	44	51	15	"	3.6	3.1	0.4
"	"	F	35	44	16	"	24.7	22.0	2.2
"	"	M	62	61	8	"	14.7	15.3	2.1

* This means that there were two experimental periods of three days.

TABLE X.—continued.

Name of Observer.	Date.	Patients'			Duration of experiment in days.	Date.	Nitrogen in faeces.		Average Nitrogen in Faeces per diem in grms.
		Sex.	Age.	Weight in Kgs.			Per cent. referred to Nitrogen intake.	Per cent. referred to total Nitrogen in Urine and Faeces.	
Markov (42)	1888	M	28	—	8	Milk only	5.1	5.6	1.2
"	"	M	29	—	8	"	6.6	6.8	1.5
"	"	M	26	—	2 x 8*	"	4.3	4.7	1.0
"	"	M	24	—	6	"	6.1	5.4	1.0
"	"	M	24	—	8	"	5.9	6.5	1.3
"	"	M	24	—	8	"	8.3	5.5	1.1
Prausnitz (43)	1889	M	—	74	3	"	6.2	7.0	1.4
Listov (37)	1892	F	21	59	2 x 8*	Milk and bread	6.3	7.4	1.3
"	"	F	24	50	2 x 8*	"	7.1	7.3	1.6
"	"	F	22	64	4 x 8*	"	6.1	7.4	1.8
"	"	F	52	48	2 x 8*	"	6.9	8.1	1.3
"	"	F	19	48	2 x 8*	"	7.2	7.4	1.7
"	"	F	19	77	2 x 3*	"	9.8	10.2	2.8
"	"	F	19	67	2 x 3*	"	4.9	4.1	0.8
Magnus Levy (40)	1893	M	16	57	3	Milk only	8.6	8.7	1.5
"	"	Same boy	24	—	8	Milk, bread, butter, sugar	9.9	11.2	0.6
Tunnicliffe & Rosenheim (61a)	1901	M	24	15	25	Milk chiefly	11.0	11.9	0.9
"	"	M	5	19	23	"	8.6	10.1	0.6
"	"	M	24	15	7	"	8.1	9.7	0.6
"	"	M	5	17	7	"

* This means that there were two experimental periods of three days.

In one case of Rudenko's, as much as 24·7 per cent. of the nitrogen of the intake was found in the fæces, but in no other case are the figures so high. Including this exceptional instance, the average results of 37 investigations upon different healthy persons having a milk diet showed that for every 100 grms. of nitrogen in the food there were 7·4 grms. of nitrogen in the fæces; and for every 100 grms. of nitrogen in the urine and fæces there were 7·3 grms. of nitrogen in the fæces, and the average daily total nitrogen in the fæces was 1·2 grms.

Camerer's experiments alone were made upon boys and girls of an age similar to that of our patient, and the figures, an average of five cases are as follows:—For every 100 grms. nitrogen in the food there were 6·1 grms. in the fæces; for every 100 grms. nitrogen in the urine and fæces there were 5·7 grms. nitrogen in the fæces; the daily average total nitrogen in the fæces being 0·6 grms.

In the next table the figures obtained upon persons suffering from different forms of Bright's disease are tabulated. In these the diet was not always milk only, though it was always light, and in some of them drugs were given (*See Table XI.*).

It will be seen that Korkounov on one occasion found as much as 74·2 per cent. of the nitrogen intake in the fæces; and Grigoriev 43·9 per cent.; but these figures are very much above the average. The lowest figures are also Grigoriev's, namely, 3 per cent. of the nitrogen intake, and 2·7 per cent. of the total nitrogen in the urine and fæces.

The average of the whole thirty-two cases shows that for every 100 grms. of nitrogen in the food, there were 14·1 grms. nitrogen in the fæces; for every 100 grms. of nitrogen in the urine and fæces, there were 14·0 grms. nitrogen in the fæces. The average daily nitrogen in the fæces being 1·9 grms.

Baginsky's patients alone approach in age to ours, and the average of his results for four observations are as follows: for every 100 grms. of nitrogen in the food there were 9·4 grms. nitrogen in the fæces; for every 100 grms. of nitrogen in the urine and fæces there were 12·0 grms. nitrogen in the fæces. The average daily nitrogen in the fæces being 1·2 grams.

TABLE XI.

Name of Observer.	Date.	Patients'			Duration of experiment in days.	Diet.	Nitrogen in faeces :—		Form of nephritis.	Average nitrogen in faeces per diem in grms.
		Sex.	Age.	Weight in kgs.			Per cent. referred to nitrogen intake.	Per cent. referred to nitrogen in urine and faeces.		
Rudenko (59)	1885	M	30	68	18	?	20.0	11.9	Diffuse nephritis	1.5
"	"	M	51	58	20	?	10.2	12.2	"	0.9
"	"	F	28	75	7	?	20.0	18.4	"	1.5
Korkounov (31)	1886	M	—	69	2 x 4	Milk	8.2	9.9	Chronic nephritis	2.0
"	"	M	—	76	4 x 8	"	74.2	46.4	"	11.6
Evdokimov (13)	1887	M	26	57	13*	Milk, Bread, Tea	21.0	23.1	Acute parenchymatous	2.5
Garine (16)	"	M	48	53	15*	Mixed	10.7	18.8	Acute interstitial	3.1
"	"	M	39	75	15*	"	31.1	39.0	"	6.2
Grigoriy (18)	1888	M	22	64	13*	Various; but chiefly milk and bread	5.0	4.2	Acute parenchymatous	0.6
"	"	M	51	81	9*	"	9.0	9.7	Chronic parenchymatous	0.3
"	"	M	27	79	40*	"	48.9	32.3	"	5.0
"	"	M	25	62	25*	Various	17.1	18.9	"	2.3
Prior (50)	1891	M	19	—	16*	"	4.6	4.7	Nephritis	0.7
"	"	M	40	—	21*	"	5.1	5.2	Bright's disease	0.7
"	"	M	57	—	16*	"	7.4	10.0	Sclerosis of kidney	0.8
"	"	M	34	—	9*	"	5.3	6.4	Chronic parenchymatous	0.9

* In these cases the experiment was not continuous, and various diets and drugs were given.

TABLE XI.—continued.

Name of Observer.	Date.	Patients'		Duration of experiment in days.	Diet.	Nitrogen in faeces :—		Form of nephritis.	Average nitrogen in faeces per diem in grms.
		Sex.	Age.			Per cent. referred to nitrogen in urine and intake.	Per cent. referred to nitrogen in urine and faeces.		
Prior (50)	1891	M	47	10*	Various	13.4	12.0	Chronic parenchymatous	1.3
"	"	M	47	19*	"	9.3	9.7	Chronic interstitial	1.0
P. Müller (45)	1891	F	25	33*	Mixed	10.9	12.9	Chronic nephritis	1.5
Van Noorden (64)	1891	F	37	5	"	16.4	22.7	Shrivelled kidney	2.9
"	"	F	27	7	"	14.6	14.1	"	1.8
"	"	F	38	9	"	4.5	5.6	"	0.6
"	"	F	43	16*	"	5.2	5.6	"	0.7
"	"	F	22	27	"	11.3	11.1	Chronic parenchymatous	1.6
Mann (41)	1892	M	45	12*	Various	19.5	26.8	"	2.1
"	"	M	53	6*	"	11.4	17.0	Sclerosis of kidneys	2.0
"	"	M	29	5*	"	8.6	10.3	"	1.5
"	"	M	49	15*	Mixed	3.1	3.6	Chronic nephritis	0.4
Kornblum (32)	1892	M	49	5	Milk	5.6	6.4	Acute nephritis	0.7
Baginsky (3a)	1893	F	13*	5	"	4.3	4.8	Scarlatinal nephritis	0.6
"	"	F	13*	28	"	10.0	14.6	Acute nephritis	1.3
"	"	F	5	6	"	17.7	21.7	"	2.1
"	"	F	12	3	Milk, Rice, Bread...			"	

* In these cases the experiment was not continuous, and various diets and drugs were given.

The table shows that very wide variations in the fæces nitrogen in Bright's disease may occur quite independently of the form of kidney lesion.

Our own figures for each day are given *in extenso* in Table I. On some of the days the patient vomited, and the nitrogen of the vomit has been added to the fæces nitrogen. The vomits were always very small, being chiefly saliva. On three days, they were estimated by Kjeldahl and found to contain 0·0482 grms.; 0·0407 grms.; and 0·0629 grms. nitrogen respectively. Being so small, the vomit was therefore on other occasions added to the daily fæces and the two estimated together.

The following is a summary of our results arranged as follows:

- (1). For successive periods of five days each, the first day being neglected.
- (2). For the period of mild uræmia.
- (3). For the different diets the patient took.
- (4). The average for the entire experiment.

TABLE XII.

	Percentage of Nitrogen in Fæces referred to Nitrogen in Food.	Percentage of Nitrogen in fæces referred to Nitrogen in Urine and Fæces.	Average Nitrogen in Fæces per diem in grms.
(1). Successive periods of 5 days—			
(a). February 20th to February 24th	13·6	14·3	1·3
(b). February 25th to March 1st	18·0	19·7	1·7
(c). March 2nd to March 6th	14·0	12·4	0·8
(d). " 7th to " 11th	20·1	20·6	1·3
(e). " 12th to " 16th	11·6	13·9	0·8
(f). " 17th to " 21st	14·0	14·7	1·3
(g). " 22nd to " 26th	11·3	13·2	1·3
(h). " 27th to " 31st	11·3	11·4	1·2
(2). Period of mild uræmia— March 2nd to March 13th (12 days)	15·9	15·7	1·0
(3). Different diets—			
(a). Milk— February 19th to March 4th) March 18th to March 22nd)	14·9	15·5	1·3
(b). Milk and bread or biscuit— March 5th to March 17th ...	14·9	16·1	1·0
(c). Milk & biscuit and Liebig— March 23rd to March 31st ...	10·9	11·8	1·2
(4). Average for entire experiment 41 days	13·9	14·7	1·2

Van Noorden and Ritter (64) concluded from their researches that the fæces nitrogen might, in Bright disease, either be almost normal or considerably higher, but that neither from the general condition of the patient, nor from the variety of the kidney lesion could it be decided beforehand whether high or normal figures would be found; and that it could not be determined whether the fæces nitrogen was due to non-absorption or to true secretion.

Our own conclusions are that, in our patient, suffering from subacute parenchymatous nephritis, and treated with purgatives:

(1). The average daily fæces nitrogen (1.2 grms.) is the same as that found as the mean of thirty-seven investigations upon healthy persons of all ages upon a milk diet.

(2). The average daily fæces nitrogen (1.2 grms.) is double that found as the mean of five investigations by Camerer upon healthy children of about the same age as our patient (0.6 grms.).

(3). The average daily fæces nitrogen (1.2 grms.) is less than that found as the mean of thirty-two investigations upon patients of all ages suffering from different kinds of Bright's disease (1.9 grms.).

(4). The average daily fæces nitrogen (1.2 grms.) is the same as the mean of four investigations by Baginsky upon children suffering from acute nephritis, at ages comparable to that of our patient.

(5). The fæces nitrogen for the whole period expressed as percentage of nitrogen in urine and fæces (14.7 per cent.) is slightly greater than that of four children with acute nephritis investigated by Baginsky (12.0 per cent.); is more than twice as much as that of thirty-seven different healthy persons upon milk diet (7.0 per cent.); and nearly three times that of five healthy children upon a milk diet, investigated by Camerer (5.7 per cent.).

(6). The influence of diet is little marked: with Liebig the proportion of fæces nitrogen (11.8 per cent.) was somewhat less than with milk alone (15.5 per cent.); or with milk and bread (16.1 per cent.).

(7). That during the uræmic period the proportion of the fæces nitrogen (15.7 per cent.) did not appreciably differ from that of the whole experiment (14.7 per cent.).

(8). The proportion of faeces nitrogen was uniformly high ; and did not at any time shew wide variations from the average.

Such high proportion, however, does not prove that the bowel was excreting nitrogen and thus doing work that would otherwise be done by the kidneys, since we have no conclusive evidence that the whole of the faeces nitrogen may not have been due to non-assimilation from the food. In our patient there were three possible sources of faeces nitrogen, namely :—

- (1). Non-assimilation of food.
- (2). True bowel excretion.
- (3). Inflammatory exudation from mucous membrane.

SECTION IX.

The Unrecovered Nitrogen.

Our patient's weight was approximately the same at the end of the experiment as at the beginning. Therefore, the loss of nitrogen (20·7 grms.) in the six weeks may be due either to experimental error, or to the sweat and shedding of hair, surface skin and psoriasis scales. How much is due to the second factor we do not know ; in spite of every care, the sum of the experimental errors of forty-one days cannot but be considerable ; because milk spilled makes the intake too high, urine spilled, the output too low.

Other workers have found wide differences. Referring only to those metabolism experiments in which the diet was milk and the person healthy, we find that Hofman (25) in a three days' observation recovered 30·3 grms. more nitrogen than was in the food ; Lapschinsky (35) at the opposite extreme, found in six days a deficit of 45 grms. Most of the experiments are for short periods only, and the variations are so great that no useful purpose would be served by giving all the results here. They have been summarised in tabular form by Atwater and Langworthy (2).

Tunnicliffe and Rosenheim's experiments upon two healthy children, whose diet was chiefly milk, are of longer duration, and comparable with our own as regards the diet and the patients' age.

The daily nitrogen deficit is tabulated in their paper (61A).

In case A, a child of $2\frac{1}{2}$ years, the deficit was 18·87 grms. in twenty-five days, with an increase of 0·17 kg. in body weight. In case B, a child of 5 years, it was 14·05 grms. in twenty-two days, the increase in weight being 0·28 kg.*

Turning now to metabolism experiments in Bright's disease, we find even wider variations. Thus, Korkounov (31) recovered an excess of 39·3 grms. nitrogen in a three days' observation in one case, while in another of the same duration his deficit was 32·4 grms.

Numerous researches, all quoted in our introduction, have been made, but for comparatively short periods continuously. Baginsky's figures alone are for children with acute nephritis on milk diet; we give them in full.

TABLE XIII.

Daily loss or gain of nitrogen in Baginsky's metabolism experiments upon children with acute nephritis. (3a.)

	A. (girl 13 $\frac{1}{2}$ years.)		B. (girl 13 $\frac{1}{2}$ years.)		C. (girl 5 years.)	
	Daily loss or gain.	Progressive deficit.	Daily loss or gain.	Progressive deficit.	Daily loss or gain.	Progressive deficit.
1st day ...	—3·043	3·043	+0·275	—0·275	—5·258	5·258
2nd " ...	—4·405	7·448	—2·280	2·005	—3·834	9·092
3rd " ...	+0·090	7·358	—2·590	4·595	—4·638	13·730
4th " ...	+2·467	4·891	—1·900	6·495	—4·679	18·409
5th " ..	—3·724	8·615	—1·220	7·715	—5·350	23·759
6th " ..	—	—	—	—	—0·831	24·590
Average daily loss }	...	1·723 No gain of weight.	...	1·543 No gain of weight.	...	4·098 0·59 kg. gained.

— Means deficit.

+ Means surplus over intake.

* In a paper published since this was written (*Journal of Hygiene*, vol. 1, No. 3, July, 1901), Tunncliffe and Rosenheim give the results of further metabolism researches upon the same children. In these experiments, which extended over twenty-eight days, the nitrogen deficits met with were:—Child A, 27·58 grms.; child B, 40·97 grms.

No experiments upon children with nephritis have extended over a longer period. None have included a period of uræmia.

Our own results both as regards the gain or loss of nitrogen each day and the progressive deficit from the beginning are given in full in Table I.

The average deficit of nitrogen per diem was 0·506 grms., which is much less than Baginsky found in his cases. An examination of the figures in conjunction with the clinical account, shows that the deficit occurred almost entirely when the general condition of the patient appeared best. When uræmic symptoms were present, the output of nitrogen almost invariably exceeded the intake. This is very clearly illustrated by the curves in the chart.

Thus, by the end of the tenth day the nitrogen deficit was 8·07 grms.; for the next twelve days the output exceeded the intake, so that on the twenty-second day the nitrogen deficit was only 2·48 grms. and the patient had gained 1·1 kg. in weight. While this recovery of nitrogen had been going on the patient was distinctly uræmic. On the twenty-third day his health improved greatly, and he continued better until the thirty-ninth day; during this time the nitrogen deficit increased to 23·75 grms. and his weight to 1·5 kg. more than at the beginning. During the last two days he became less well, and again excreted more nitrogen than he took as food, so that the deficit fell to 20·75 grms. This was the commencement of a second uræmic period. Unfortunately we were unable to continue the work after March 31st, but we saw the patient the following week and he was distinctly uræmic.

We had intended to compare the nitrogen deficit for the period (Feb. 19th to March 6th inclusive) when baths were given, with that of the remaining time without baths. The average daily deficit for the period of baths was 0·23 grms. nitrogen; for the period of no baths was 0·68 grms. nitrogen. At first sight this seems to shew less loss of nitrogen by the skin when baths were given than without them; but the figures will not bear this interpretation for the following reasons:—

- (1). The body weight did not remain constant.

(2). The amount of œdema was variable and we had no means of measuring it exactly and so determining how much of the variation in body weight was due to water.

(3). The period of uræmia supervened.

Tunnicliffe and Rosenheim (61A) in their experiments on two healthy children found an average daily deficit of 0.75 and 0.64 grms. nitrogen. There were, it is true, gains of weight of 0.17 and 0.28 kg. respectively, which, if entirely due to proteid, would account for part of the deficit.

Supposing that every 100 grms. of increased body weight meant 3.3 grms. of food nitrogen converted into living tissues, there would still be 0.53 and 0.22 grms. nitrogen daily unaccounted for. We will compare these figures with our own. Over the whole experiment, we found an average deficit of 0.50 grms. nitrogen daily.

This is less than the average figures in Tunnicliffe and Rosenheim's cases, if no account be taken of altered body weight. In our case, the presence of a varying œdema vitiates allowances for changes in weight. An increase in our patients' weight was certainly not all due to building up of nitrogen into living proteid.

For example, at the end of the twenty-second day the total deficit was 2.484 grms. nitrogen; but the body weight had increased by 0.85 kg. If the latter were entirely due to proteid built up, it would contain 27.05 grms. nitrogen; so that 18.9 times our nitrogen deficit would be accounted for.

This is not probable. Even though we make no allowance for altered body weight in our case, and yet concede that the gain in weight in the healthy children was entirely due to proteid, we find that our daily nitrogen deficit was less than Tunnicliffe and Rosenheim's in their longest experiment, though greater than that in their other. Remembering that our patient was covered with psoriasis, and that during the period when baths were given he often perspired very freely, whilst urea has been found in the sweat of uræmic patients (61), we expected our deficit to be greater.

The conclusions which we draw from this section of our work are therefore two, namely, that in our patient suffering from sub-acute nephritis :—

(1). The uræmic period was marked, not by a retention of nitrogen, but by an output in the urine and fæces of more nitrogen than was taken in the food, the reverse being true when he was in better health.

(2). The excretion of nitrogen by paths other than the kidney and bowel was not appreciably greater than it is in health.

SECTION X.

General Conclusions.

Our general conclusions are: That, in our patient, with subacute parenchymatous nephritis :—

(1). The proportion of nitrogen excreted as urea was not less than that found in healthy persons, if the coagulable proteid in the urine be regarded as a foreign constituent, and not as a true excretory product.

If the coagulable proteid nitrogen be included, the proportion of urea nitrogen was less than in health.

(2). The above statements held good both for the uræmic and for the non-uræmic periods.

(3). The uræmic period was marked by :

(a). An output of nitrogen in excess of the intake.

(b). A high, and almost constant, ratio $\frac{\text{uric acid}}{\text{urea}}$.

(4). The fæces nitrogen constituted a larger proportion of nitrogen output than has been found in healthy children on a milk diet.

(5). The loss of nitrogen by other channels than the urine and fæces was not apparently greater than in health.

We desire to express our gratitude to Dr. Spriggs for his advice, and especially for his assistance during the illness of one of us. We thank Dr. Taylor and Dr. Fawcett for permission to investigate

the case; Dr. Pembrey for the facilities he gave us in his laboratory; and the Matron of Guy's Hospital, Sister Clinical and her Nurses, without whose hearty co-operation the work could not have been accomplished.

The expenses of the research were defrayed out of a grant from the Scientific Grants Committee of the British Medical Association to Dr. Pembrey.

SECTION XI.

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ROENTGEN-RAY DIAGNOSIS OF RENAL CALCULUS.

By E. W. H. SHENTON.

RADIOGRAPHER TO GUY'S HOSPITAL.

THE detection of renal calculus, at once the most difficult and most useful branch of Roentgen-ray science, has not received the amount of attention that its merits deserve.

This can be accounted for in two ways, firstly by the dread of dermatitis, shared alike by patient and medical adviser, secondly, by the disappointment arising from a haphazard way of taking a skiagram of an abdomen and calling it an X-ray examination of that part. The novice with improper apparatus is responsible for the dermatitis in these days, and there are few branches of medicine in which this well-meaning but incompetent person would not be dangerous. That dermatitis is an accident, and one that can and should be avoided, is proved by the records of the Guy's Hospital department, for during the years that this work has been carried on there has not been a single instance of inflammatory reaction taking place after a photographic exposure. Even in cases where the rays have been used for therapeutic purposes, and exposures of ten minutes several times a week have been given, there is not a single recorded instance of dermatitis. Most examples of Roentgen-ray dermatitis, if the history be carefully enquired into, prove to be due to want of knowledge on the part of those utilizing the rays.

The unsatisfactory results from carelessly taken skiagrams will always be present, unless the fact becomes more generally known that a renal examination with the X-rays is not merely "photographing a patient's kidneys," a foolish and prevalent expression; prevalent because of the want of knowledge generally among the profession of things pertaining to the rays; foolish because it is rare in the skiagram of an abdomen to see the kidneys, for, as a rule, the less that is seen of the kidneys the more chance there will be of seeing a calculus. It is certainly time that these fallacious ideas were dispelled and a fair statement of Roentgen renal examination set forward, with its successes and failures. Supposing, as it is fair to do, that dermatitis is a preventable accident, there is surely no more harmless system of examining the abdomen than that afforded by Roentgen's discovery. A skiagram *per se* is unreliable in all but the most obvious cases and, as in other branches of diagnosis, it is only by a systematic routine of examination that the percentage of failures can be reduced to a minimum. Mr. Jonathan Hutchinson, in a recent paper upon renal subjects, in which he deals with the uses of the X-rays in calculus cases, merely speaks of the photographic aspect of the subject, indeed he does not even state the possibility of the screen being of service. That photography alone is likely to prove very unsatisfactory, can be abundantly proved, for it is possible to so arrange tube and patient, that an obvious calculus does not show in a photographic plate, and this is the error that care alone can exclude, and it is also possible for a calculus to move so much with respiration that it leaves no trace upon the plate.

To merely speak, then, of the photographic aspect of kidney examination is to leave out perhaps the most valuable part of the system. Examination for renal calculus must be divided into two distinct parts, the fluoroscopic and the radiographic; what is seen upon the screen and what is discoverable upon the skiagram. As in other diagnostic methods, it is of extreme importance to have a confirmatory test, and radiography is usually the confirmatory test of radioscopy. The operator

has first to make himself familiar with the radiosopic appearance of the normal abdomen, before attempting to give an opinion on a renal case. In this he will meet with many difficulties. The various effects produced by changes in the position of the tube are mystifying (to obviate this difficulty it has been suggested that the tube should always be put opposite a certain spot. This suggestion is ridiculous, as it would take away at once the chances of finding a stone when not situated within a small area). Then, again, the shadows of vertebræ and other opaque objects are at the best so very faint that a considerable time must be expended before the organs which they represent can be recognised with any certainty. One might draw a parallel between these faint shadows and the faint sounds heard in a stethoscope. To the untrained eye or ear neither have significance, yet it is possible that a careful study of these ghostly screen effects may lead to a more delicate and perfect method of ascertaining the condition of the abdominal organs, as a proper appreciation of the faint auscultatory sounds have led to the perfection of chest diagnosis. However this may be, the search for calculus is an established and comparatively simple process, and one which, whenever accuracy of diagnosis is of moment, should never be omitted. The observer has not to trouble about any refinements in the quality of the shadow, but merely to convince himself that a shadow of an abnormal constituent of the abdomen is present, and by its size and position and the compatability of the symptoms to decide whether this shadow is that of a calculus. By considering the symptoms it will be at times sufficient to examine the patient but once, for a good history and an obvious foreign body in the particular renal region to which these belong, will be sufficient evidence; but in the event of a very doubtful history and an indistinct shadow, possibly caused by some intestinal contents, it will be most advisable to examine again at an interval that will have allowed such foreign matter to pass away.

It will be asked in what percentage of cases examined are the rays successfully used both as positive and negative evidence. This is a very pertinent question and one about which observers

differ. Some X-ray workers naturally see much better than others in an almost darkened room, and these people, as one would expect, see more upon the screen. Others, who are not so successful in the screen work, will read the skiagram with a degree of ease that will appear astonishing. Again, one operator will know the degree of penetration requisite in his tube to suit his patient, and in consequence the percentage of his failures will be lessened. Another will know how to make up in exposure what he is unable to get in the penetrative quality of his tube. It is, therefore, much more the personal equation than any fixed standard of excellence in the apparatus that has to be considered.

I have come across some quite busy X-ray workers who admit that they have never been able to demonstrate the presence of a calculus upon screen or skiagram. This may at times be due to inferior apparatus, but as often as not the cause is want of observation. Speaking of the cases seen in the Guy's Hospital radiographic department in the past two years, I can give details which, without being regarded as statistics, will help the reader to form his own opinion of the usefulness of this method of renal exploration. It is most probable that with the improvements of the future the percentage of failures will diminish, and certainly the past year has shown a distinct improvement on the year before, only three mistakes having been made against five in the previous twelve months, although during the latter period many more cases were examined. It is unlikely that absolute accuracy will ever be obtained in this as in any other method of diagnosis. Mention will only be made of the work done in the past two years, for it is only in this period that the Roentgen light has been produced in sufficient quantity to be thoroughly useful. There have been cases in which the rays have shewn calculi to be present where the clinical signs did not more than suggest the possibility of this condition. There have been others where the patients have had no symptoms that would in the least have suggested calculus, and the examination has been almost accidental. There have been cases, perhaps, more remarkable, where the symptoms have been so strongly pointing to calculus, that the exploration with the rays has been considered a waste of time,

and yet neither the rays nor the surgeon have been able to bring to view a particle of calculus. There have been cases in which the rays have shewn calculus, but the surgeon has not. The following is a summary of the results obtained.

Cases examined, two hundred.

Cases in which the rays and surgeon found calculi, twenty-eight.

Cases in which surgeon found calculi but the rays not, eight.

Cases stated not to have calculi by the rays, and operated on with negative results, eleven.

Number of cases in which the rays found calculi, but surgeon did not, two.

Therefore, the number of cases in which the result obtained by the rays has been proved to be correct amount to thirty-nine.

The instances in which they have been proved wrong are eight. The one hundred and fifty-three remaining cases are doubtful as they have not been operated upon, but in most instances the negative evidence of the rays has been confirmed by subsequent history.

It will be seen, therefore, that the positive evidence is almost perfectly reliable, the negative not absolutely, but should be allowed to have weight when considered with other symptoms.

The errors occurred in stout people and in those who presented abnormal opacity to the rays, or in cases where the stones were very small, or where composed of uric acid or urates without admixture of more opaque salts. Fig. 1 shows the relative densities to the rays of the three most common salts.

The method of examination has been as follows:—

Position of the patient with relation to the tube, etc.—There are several reasons why the horizontal position of the patient is best for abdominal examination. The tube should be beneath and the screen above. The couch, the height of an ordinary table, should have an X-ray transparent top of sailcloth. When the patient lies upon his back the antero-posterior measurement of the abdominal region are considerably reduced. If he lies upon his face there is a still greater diminution of the depth, and this reduction is in the highest degree important. The abdominal

region is so dense to the rays that every effort has to be made to lessen this density. Having turned the patient upon his face he should be told to place his arms above his head and lie as flat as possible. It is as well to allow no pillow for the head, as this does not add to the comfort and prevents the back from being straightened. Every attempt should be made to get the lumbar spine straight, even to the extent of placing a pillow beneath the abdomen, care being taken to see that there is nothing in the pillow to interfere with the transit of the rays. The tube, which in the couch that I have specially designed for this work can be moved by the operator into any position while the screen effect is watched, is now brought to within about six inches of the patient's abdomen as he lies upon his face. This measurement is only approximate and will vary with the condition of the tube and the size of the patient. The screen is placed upon the patient's back. By this means it is impossible for the patient to move into contact with the tube and the chance of accidental shock is eliminated. The current is now passed through the tube and the effect upon the screen noticed. It is as well to place the tube at the commencement of the examination beneath the upper part of the trunk to note how well the chest, a more transparent region, is shewn upon the screen. Then as the tube is gradually moved downwards careful scrutiny can be made of the abdominal contents in the order of their appearance. The various changes in the effect as seen while moving the tube from the chest down toward the pelvis are shown in the illustrations. Figs. 2, 3 and 4.

The organs which are at times visible are :—

Liver. Upper margin well defined, the lower seldom seen clearly. Fig. 5.

Spleen at times seen, as in Fig. 5.

Stomach. Evident occasionally as a clearly outlined transparent area. It is best seen when distended with gas. (Figs. 22 and 23).

Large intestine. This shews well when distended with gas. The sacculated form is quite easily made out. (Fig. 23).

The small intestine is hardly ever visible.

Kidneys. These are at times visible when the surrounding organs are filled with gas. The lower borders are usually the only parts that can be made out (Fig. 29). The more flatulent the patient, the more can be made out of the abdominal contents, and for purposes of examining a stomach for chronic dilatation the rays are extremely useful if means are adopted to procure the gaseous distention of this viscus.

A calculus must be sought with the utmost care, and to do this the tube must be moved continually. When any suspiciously opaque mass comes into the field, it should be viewed from all directions, and the tube stationed in that position in which the object is the most visible, and a plate laid face downward upon this part of the patient's back.

During exposure, which, if the tube used is the right one, should not exceed a minute, the screen can be laid upon the plate and the picture watched. This will at once tell the operator whether he has selected the right place, for it is wonderfully easy to place the plate incorrectly.

It will be noticed in the examples of renal stone here depicted (Figs. 6 to 22), that the tube must have been situated differently for nearly every case; there are several examples where, had the tube been otherwise placed, no calculus would have been visible. These alone point to the importance of careful screen work. There have even been cases where a stone has been clearly seen upon the screen, and owing to respiration no photographic record could be obtained. In conclusion, it is well to warn the reader against the skiagram that is too good. The clearly defined spine, darkly pictured upon an almost white background is, perhaps, the least likely of all to show a renal calculus. The renal regions should be grey, and show the utmost detail.



Roentgen-ray Diagnosis of Renal Calculus.

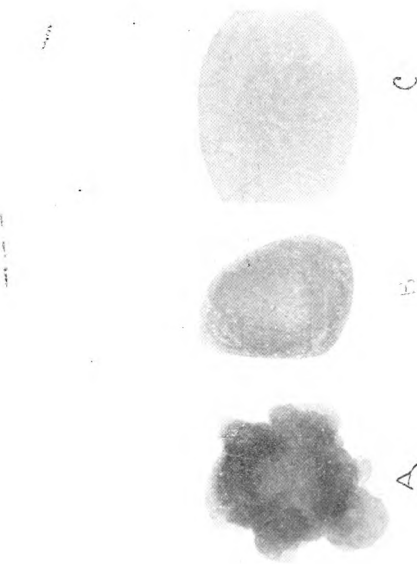


FIG 1.—An X-ray photograph of the three principal varieties of calculi—
a. Oxalate of lime calculus. *b.* Phosphate calculus.
c. Uric acid calculus.

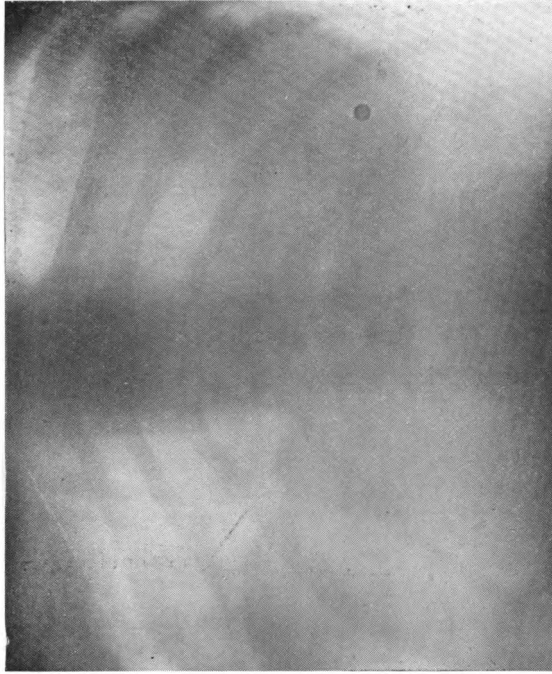


FIG. 2.—Showing the effect of placing the tube behind the upper part of the abdomen.

Röntgen-ray Diagnosis of Renal Calculus.



FIG. 3.—Showing the effect of placing the tube opposite the umbilicus.

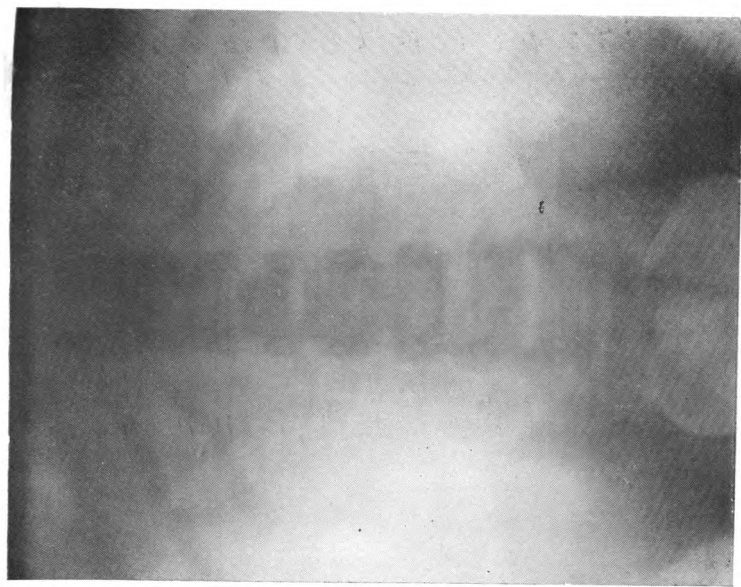


FIG. 4.—Showing the effect when the tube is placed behind the lower part of the abdomen.

Roentgen-ray Diagnosis of Renal Calculus.

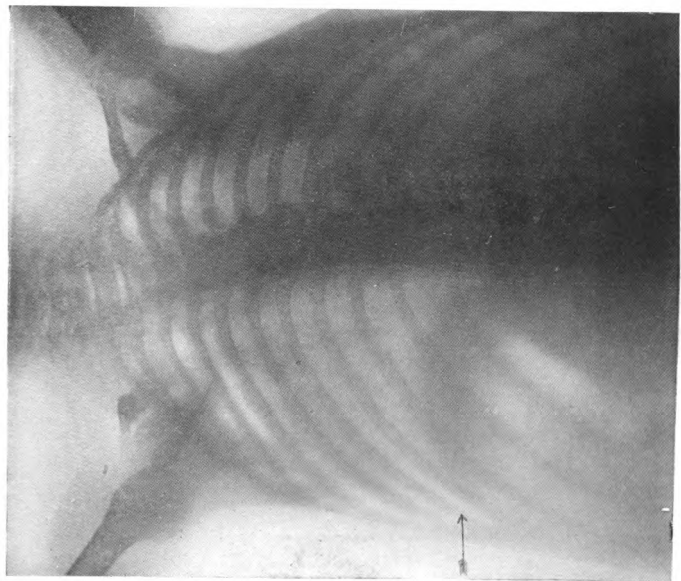


FIG. 5.—Showing the spleen.

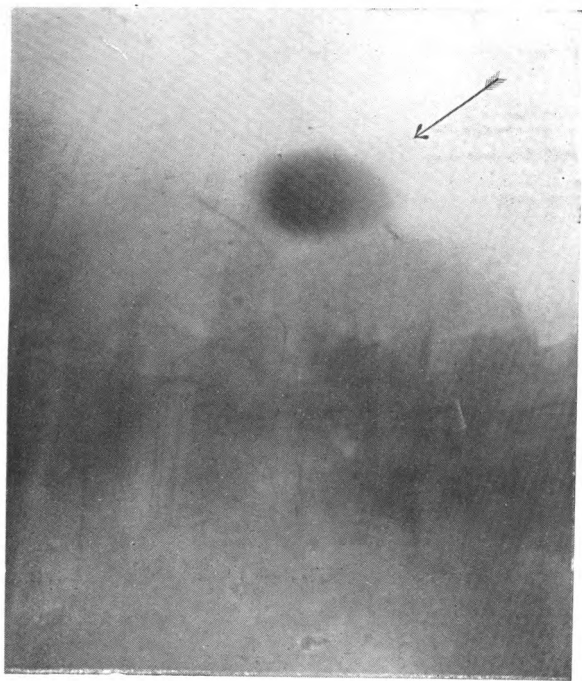


FIG. 6.—A large calculus in the right kidney, composed of oxalates and phosphates. Exposure 20 secs.

Roentgen-ray Diagnosis of Renal Calculus.

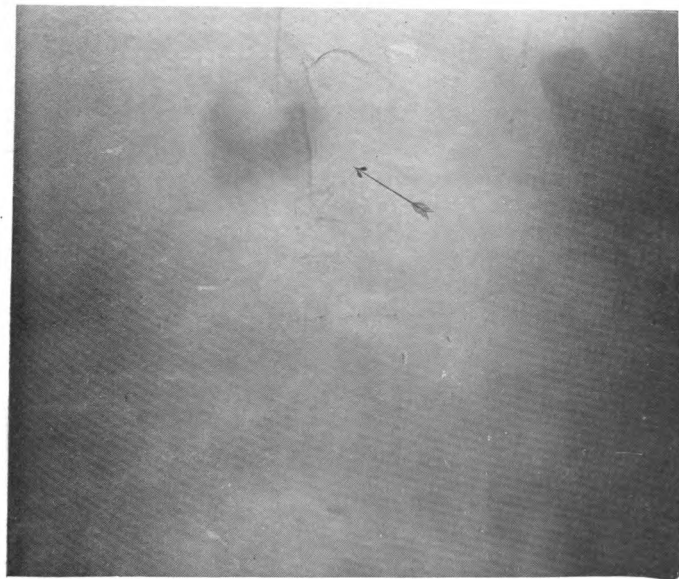


FIG. 7.—A phosphatic calculus in the left kidney.
Exposure 18 secs.

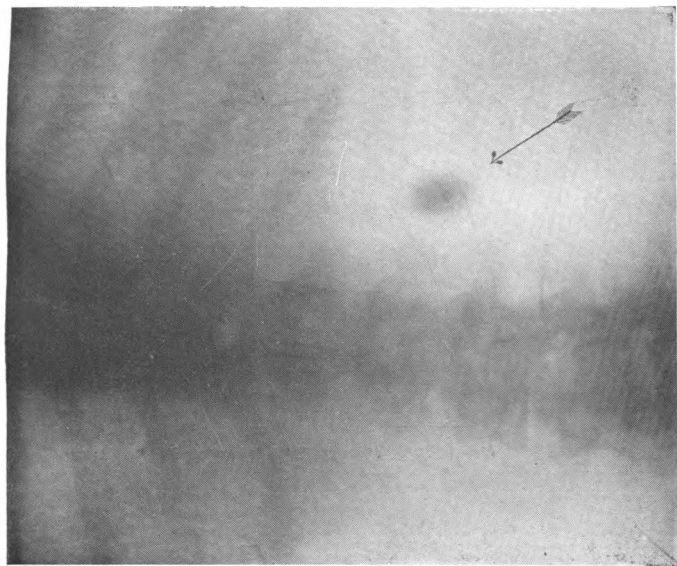


FIG. 8.—Oxalate of calcium calculus in the right kidney.
Exposure 20 secs.



Roentgen-ray Diagnosis of Renal Calculus.

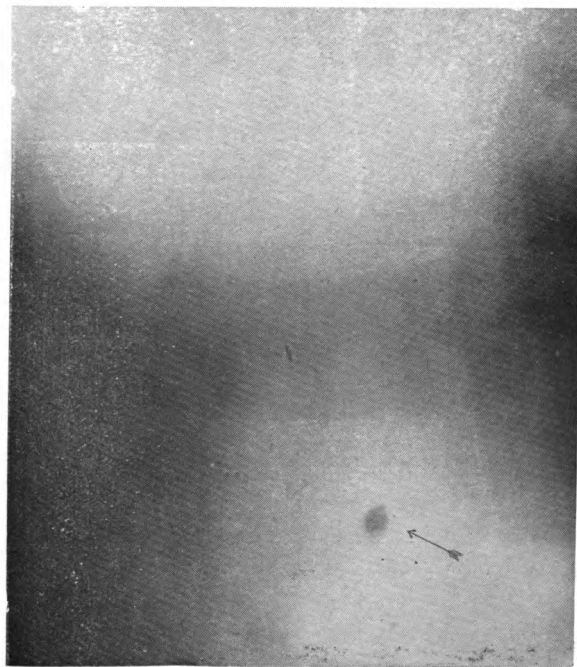


FIG. 9.—A small phosphatic calculus in the left kidney.
Exposure 20 secs.

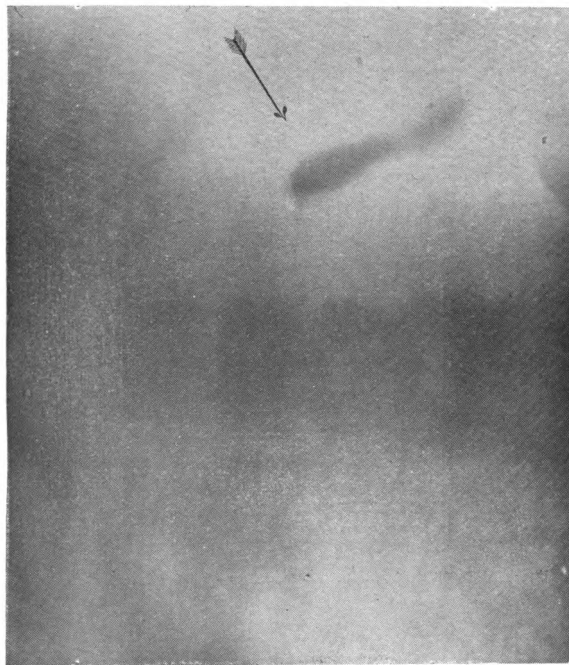


FIG. 10.—A calculus in the right kidney.
Exposure 20 secs.



Roentgen-ray Diagnosis of Renal Calculus.

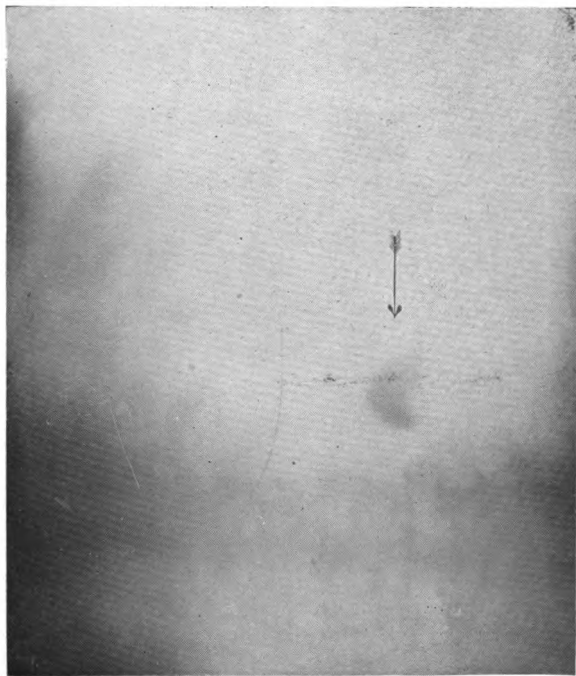


FIG. 11.—A calculus in the right kidney.
Exposure 30 secs.



FIG. 12.—Three calculi in the right kidney.
Exposure 30 secs.

Roentgen-ray Diagnosis of Renal Calculus.

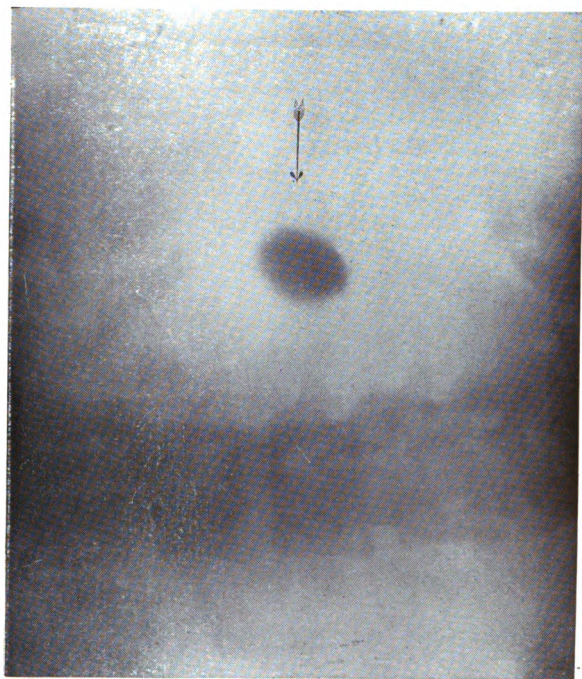


FIG. 13.—An oxalate of calcium calculus in the right kidney.
Exposure 20 secs.

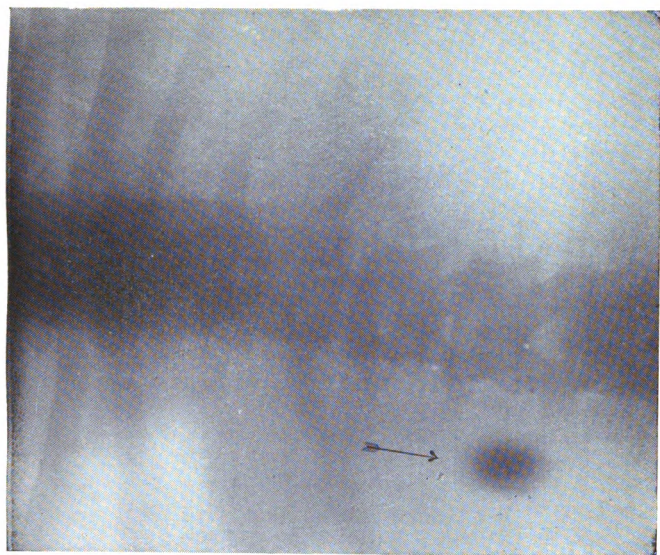


FIG. 14.—An oxalate of calcium calculus in the left kidney.
Exposure 12 secs.



Roentgen-ray Diagnosis of Renal Calculus.



FIG. 15.—A phosphatic calculus in the left kidney.
Exposure 20 secs.

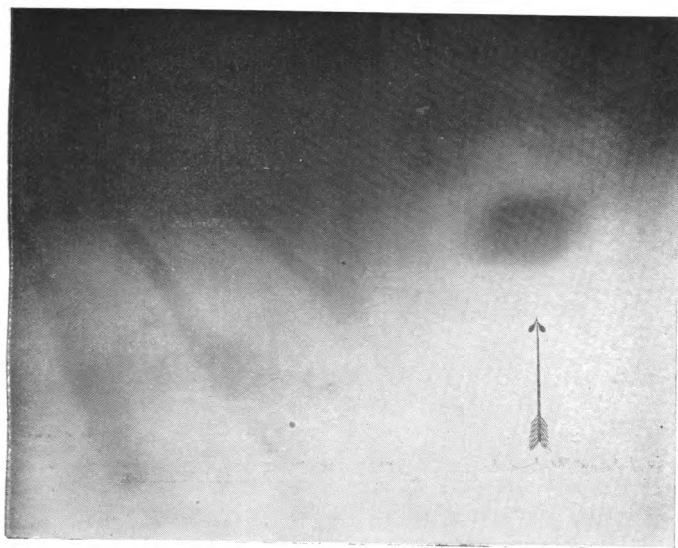


FIG. 16.—Seven calculi in the left kidney, composed of phosphates and urates. Exposure 30 secs.

Roentgen-ray Diagnosis of Renal Calculus.



FIG. 17.—A calculus in the left kidney.
Exposure 30 secs.



FIG. 18.—A calculus in the right kidney.
Exposure 20 secs.

Roentgen-ray Diagnosis of Renal Calculus.

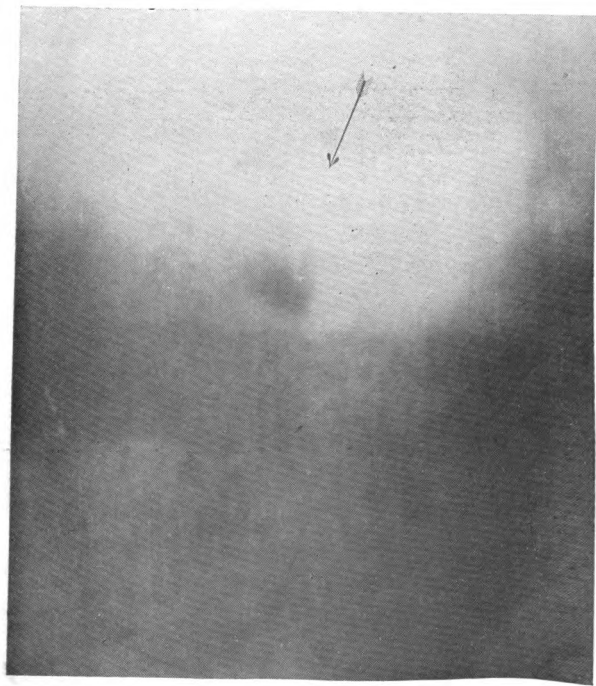


FIG. 19.—A calculus in the right kidney, composed of uric acid.
Exposure 30 secs.



FIG. 20.—Two small calculi in the left kidney.
Exposure 12 secs.



Röntgen-ray Diagnosis of Renal Calculus.

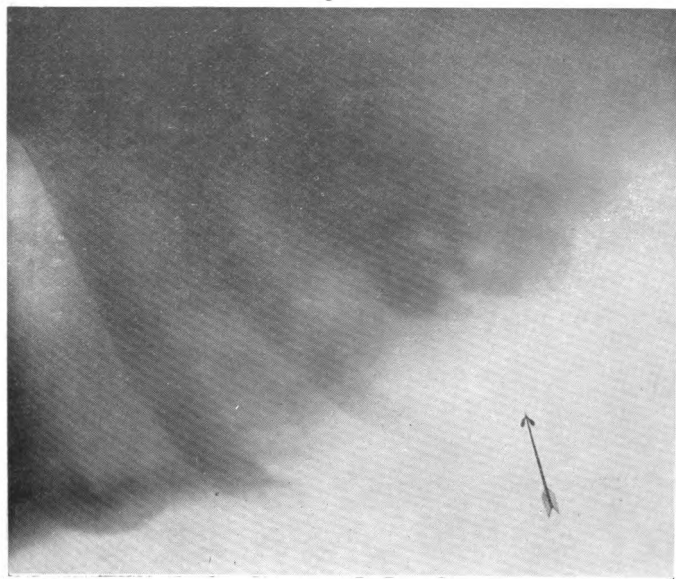
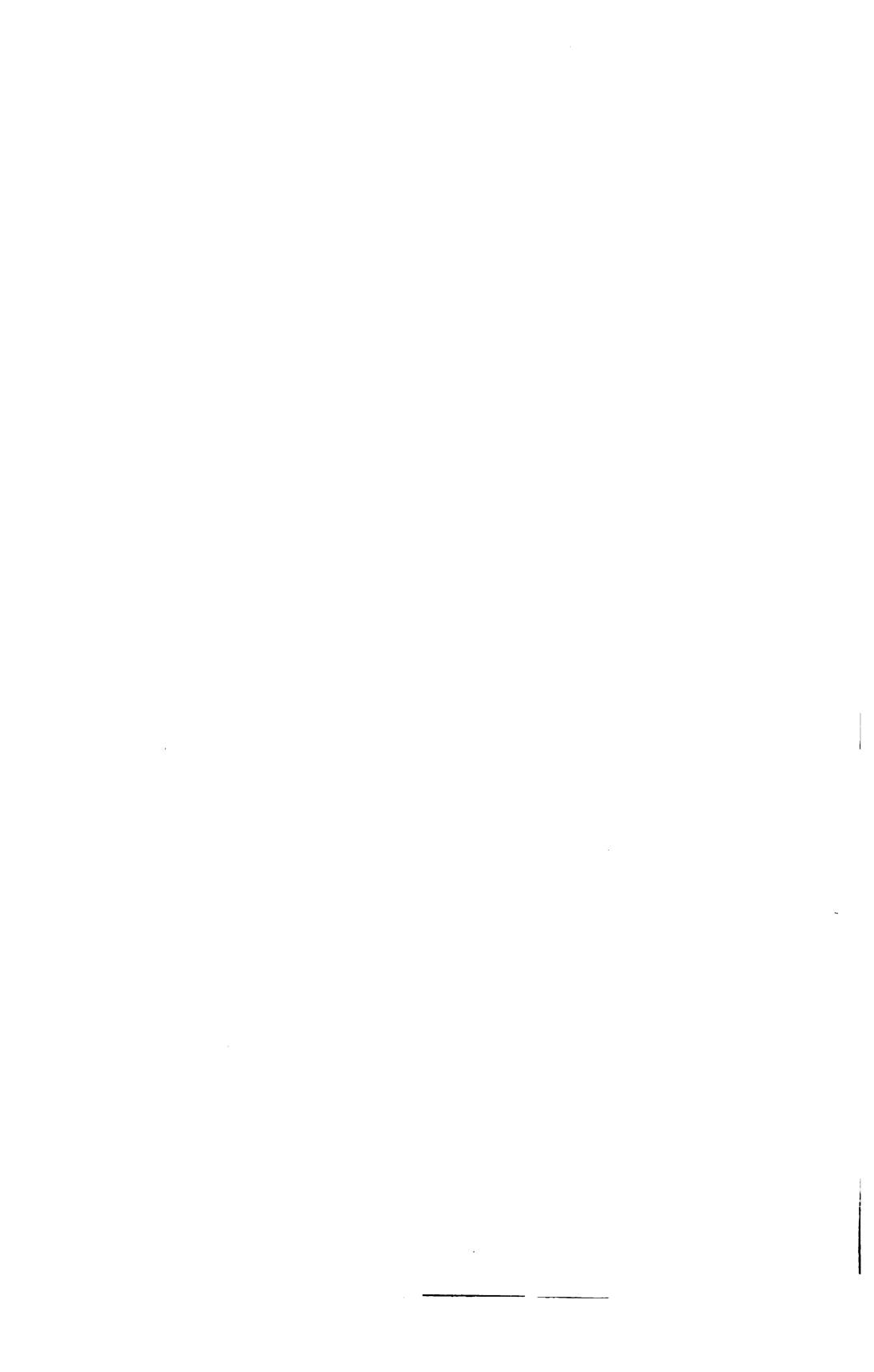


FIG. 21.—Several uric acid and phosphatic calculi in the left kidney. Exposure 20 secs.



FIG. 22.—A calculus in the left kidney situated immediately below a distended stomach. Exposure 20 secs.



Röntgen-ray Diagnosis of Renal Calculus.

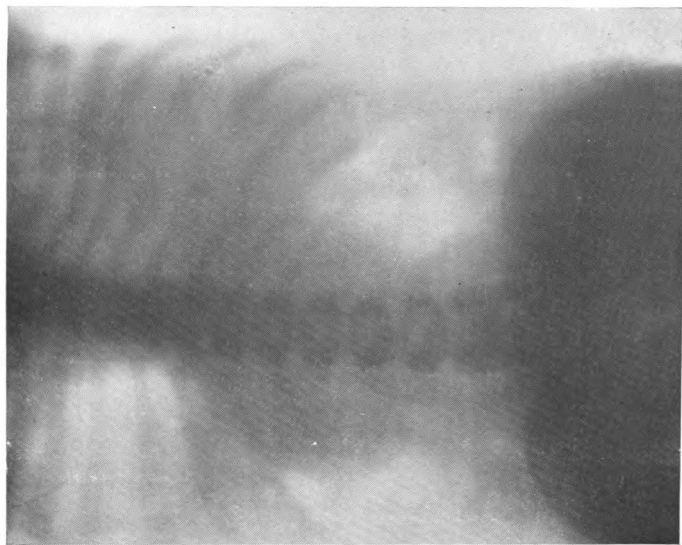
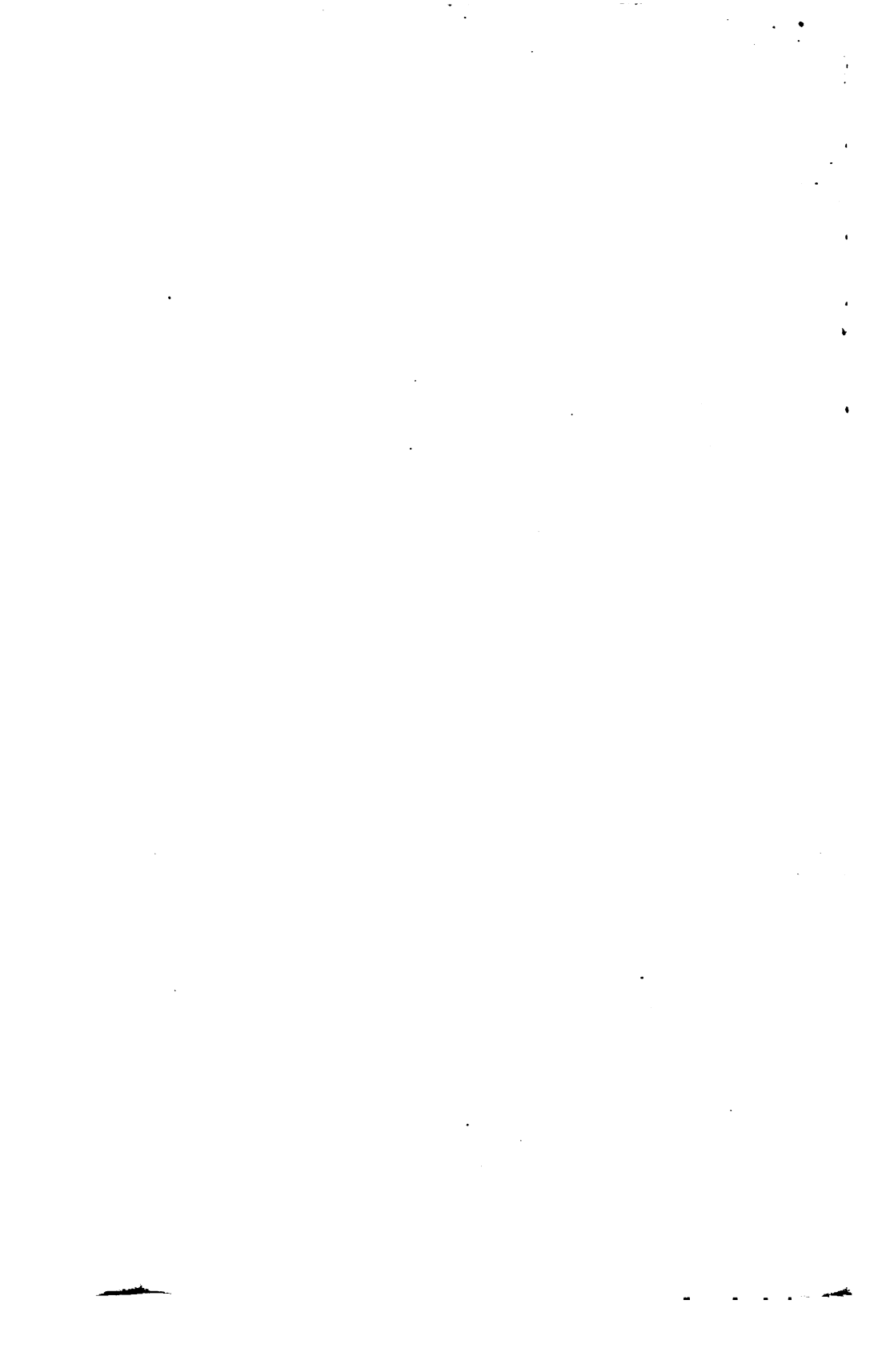


FIG. 23.—Example of a skiagram strongly supporting the negative evidence obtained by screening. Exposure 15 secs. In this the colon is shown by the light areas on either side. The stomach by the light oval patch at the left hand top corner of the picture. The dark patches on either side of the spine represent kidneys which are only evident because of the flatulent distension of the intestine.



BACTERIA IN THROMBI.*

By J. H. BRYANT, M.D.

ASSISTANT PHYSICIAN TO GUY'S HOSPITAL.

DURING the last few years considerable attention has been drawn to the influence of micro-organisms in the causation of thrombosis by the works of Cornil, Widal, Vaquez, Welch, Flexner, Pakes and others. Many thrombi, which formerly would have been classed as marantic, must now, in the light of careful bacteriological investigations, be looked upon as infective. Vaquez, in his work on phlebitis, draws attention to the presence of bacteria in these so called marantic thrombi and in the adjacent parts of the vessel walls, and looks upon the cause of the thrombosis as a primary phlebitis. One of the following cases appears to bear out this view, the chronic ulcers on the legs affording a ready access for the bacilli, and well accounting for a primary phlebitis of the adjacent veins. The following is an account of the cases:—

CASE 1.—Wm. W. was admitted on January 1st, 1900, under the care of Dr. Hale White, for dyspnœa, cough, and œdema of the legs. He was found to be suffering from mitral stenosis and regurgitation, and tricuspid regurgitation. On May 23rd, he became very cyanosed, gasped for breath, and had some hæmoptysis. He picked up a little after this, but died suddenly on June 1st.

* A paper read before the Pathological Society of London at the Laboratory Meeting held at Guy's Hospital on May 7th, 1901.

The post-mortem examination was made five hours after death. The body, legs and feet were markedly œdematous, and there was a large unhealthy looking ulcer on the outer surface of the upper third of the right thigh. The lungs were tough. On section it was found that all the branches of the pulmonary arteries were thickened, dilated and atheromatous. The branches of the pulmonary artery in the lower lobes of both lungs were filled with ante-mortem thrombi, which could be traced to the two main branches, where they united and formed a large thrombus, which filled the pulmonary artery itself, the end of the thrombus being just above the pulmonary valves. There were several recent infarcts in both lower lobes. The heart weighed 542 grammes. The right ventricle was considerably hypertrophied. The mitral orifice was much stenosed, its circumference being about twenty millimetres. The lower third of the abdominal aorta was also blocked by an old ante-mortem thrombus, which extended to the iliac arteries, both of which were also thrombosed. The pulmonary thrombosis had the appearance of having commenced in the smaller branches, and of having gradually extended upwards to the main branches.

The outside of one of the main branches of the pulmonary artery was carefully sterilized, and cut open with a sterilized scalpel. Cultures and cover glass preparations were made from the centre of the thrombus. The microscopical preparations showed streptococci and staphylococci. The cultures also showed streptococci and staphylococci. The staphylococcus was identified by its cultural reactions as the *staphylococcus albus*.

CASE 2.—Emma G., æt. 40, was admitted under the care of Dr. Newton Pitt, into Mary ward, Guy's Hospital, on March 22nd, 1900. She was suffering from œdema of the right leg. The right internal saphenous vein was thrombosed and felt hard and tender. She made good progress until April 26th, when diarrhœa and vomiting commenced. She gradually became weaker and died on June 6th at 8.30 a.m.

The post-mortem examination was made twenty hours after death. The right leg was œdematous. The right common iliac vein and the lower part of the inferior vena cava were filled with an old

thrombus which completely blocked the former. The right internal saphenous and femoral veins were also thrombosed. There was well-marked tuberculous peritonitis and perihepatitis. The iliac vein was ligatured in two places and then removed. The outer surface of the vein was carefully sterilized, and cut open with a sterilized scapel. Cultures on various media and microscopical preparations were made from the centre of the thrombus. The cover glass preparations showed a few bacilli and a (?) diplococcus. One organism only was obtained from the cultivations. It gave the following reactions. The organism was a bacillus which was fairly motile and varied considerably in length. It stained well with methylene blue, but did not retain the stain after treatment by Gram's method.

Cultures.

Broth.—In twenty-four hours the broth became quite turbid and after six days gave an indol reaction.

Agar.—A thick, moist, profuse, slimy, translucent growth, spreading all over the surface. It had a curious dull metallic appearance.

Litmus Milk.—There was a distinct acid reaction at the end of twenty-four hours. On the third day some coagulation had taken place.

Urine.—Was rendered universally turbid in twenty-four hours, and there was a crystalline deposit. It had a strong alkaline reaction. A microscopical examination of the crystals showed them to be triple phosphates.

Potato.—After forty-eight hours there was a buff-coloured, shiny-looking growth.

Gelatin.—In the shake culture there was abundant gas-formation and liquefaction.

Blood-serum.—A moist grey growth which was followed by liquefaction of the medium.

The organisms grew anærobically. It was identified as the *proteus vulgaris*.

CASE 3.—Joseph L., æt. 54, was admitted into Stephen ward, Guy's Hospital, under my care on September 22nd, 1900, for dyspnœa and swelling of the abdomen and legs. At the age of

twenty-four he was severely kicked on the left leg, and an ulcer resulted which had persisted and was present when he was admitted. He had first noticed the dyspnoea on exertion at the beginning of August and about this time his abdomen began to enlarge. Two days after the abdominal enlargement was noticed his left leg began to increase in size, this date probably indicating the time at which the veins became thrombosed.

On admission, he was found to be suffering from ascites. Both legs were swollen and oedematous the left much more so than the right. Both saphenous veins could be felt to be thrombosed. A large chronic ulcer was present on the inner side of the lower part of each leg and there was a good deal of pigmentary change in the adjacent skin. He gradually wasted and became worse. On November 13th, paracentesis abdominis was performed and some blood-stained fluid was withdrawn. He suddenly became worse and died on the following day at 11 a.m.

The post-mortem examination was made four hours after death. Diffuse carcinoma of the stomach with secondary growths in the pleura, pericardium, and peritoneum were found. Both saphenous, femoral and iliac veins were thrombosed.

Microscopical preparations and cultures on broth and agar were made from the left saphenous vein. The skin was sterilized for some distance over the inner side of the thigh and was then incised with a sterilized scalpel. The vein was exposed by a dissection with sterilized instruments. The surface of the vein was carefully sterilized and incised with a sterilized knife. The microscopical preparations and cultures were made from the centre of the thrombus. A portion of the right saphenous vein was ligatured and removed intact. The outside of the vein was then carefully sterilised and cut open and similar preparations were made.

The microscopical preparations from the left saphenous vein showed a few rather long bacilli which retained the stain well after treatment by Gram's method.

The cultivations from this vein showed only one organism. The bacillus was large and was distinctly motile. It stained

well with carbol methylene blue and retained the stain after being treated by Gram's method.

The Cultures.

Broth.—Was rendered turbid in twenty-four hours and the surface was covered with a distinct scum.

Agar.—A rapid, dry, yellowish, spreading growth with a markedly wrinkled surface.

Litmus milk.—Very little change in forty-eight hours. At the end of four or five days there was still a blue colour and no definite clotting was noticed.

Gelatin.—The stab culture showed at the end of forty-eight hours considerable liquefaction at the surface, with a pale yellowish growth in the depth. On the fourth day the top of the liquefied gelatine was covered with a white scum.

The slant culture showed extensive growth and liquefaction at the end of forty-eight hours.

Potato.—A profuse raised brownish growth with a wrinkled surface.

The organisms also grew well under anærobic conditions. The organism was identified as the bacillus subtilis. The microscopical preparations from the right saphenous vein showed cocci. No bacillus was found.

Cultivations showed only one organism. The organism was a coccus which stained well with methylene blue and retained the stain after treatment by Gram's method. It gave the cultured reactions of the staphylococcus albus.

It will be seen from the above that the bacillus proteus vulgaris in pure culture was obtained from Case 2, the bacillus subtilis in pure culture from the thrombosed left saphenous vein, and the staphylococcus albus from the thrombosed right saphenous vein in Case 3, and staphylococci and streptococci from Case 1.

These results confirm the observations of other workers in this field, that bacteria are found not only in thrombi of infective diseases, but also in those which occur in cachectic and anæmic conditions and in cardiac affections. In the thrombi which occur as complications of diseases which are due to a recognised micro-organism, it by no means follows that the specific micro-organism

of that disease will be found, for, frequently the presence of some other organism, and especially the streptococcus pyogenes and the bacillus coli communis may be demonstrated, indicating the probability of a secondary infection.

A point of very great interest is the occurrence of not only these well-known pathogenic bacteria, but also of organisms which are usually looked upon as non-pathogenic. The question naturally arises as to the relation these non-pathogenic micro-organisms have to the thrombosis. Mr. Pakes has recently reported to the Pathological Society a case of venous thrombus, in which the bacillus proteus vulgaris was found in pure culture. He was of opinion that there was an important causal relationship between the bacillus and the thrombosis.

The possibility of accidental contamination may be raised as an explanation of the presence of the bacillus subtilis, and possibly of the proteus vulgaris in the thrombi. The most stringent precautions were, however, taken to prevent the possibility of an accidental external contamination; and, further, the bacillus subtilis has been found in the organs of the body after death by other observers in cases where a post-mortem invasion was unlikely.

Taking into consideration the precautions which were taken to prevent the possibility of external contamination when the cultures were made, and the periods after death when the post-mortem examinations were made, viz., five hours after death in the first case and four hours after death in the third case, the possibility of an external contamination or a post-mortem invasion is improbable.

The mere presence of an organism in a thrombus does not necessarily imply that it was the cause of the thrombosis. Slowing of the circulation, an altered condition of the blood, such as is associated with anæmic and wasting conditions, and the presence of toxines, may all predispose to thrombosis; the bactericidal power of the blood is probably much reduced under such conditions, so that non-pathogenic organisms may flourish and become relatively pathogenic. On the other hand, micro-organisms which are found in thrombi may be found in the blood in cases in which there are no thrombi in any part of the body; for

example, I have obtained pure cultures of streptococci from the blood in cases of infective endocarditis, puerperal septicæmia, pyæmia, and diabetic coma; pneumococci from a case of general pneumococcal infection, and the bacillus coli communis from a case of typhoid fever, etc.

In the case of the pulmonary thrombus, which contained staphylococci and streptococci, there must have been several factors leading to the thrombosis, viz., slowing of the blood-stream, which was the primary state, an alteration of the condition of the intima of the blood-vessels, and the presence of the micro-organisms. That there was an ante-mortem invasion by these micro-organisms I have no doubt, but whether it was the actual determining cause of the thrombosis or whether the thrombus was secondarily infected, I must leave an open question. These organisms probably gained access to the blood through the lungs.

With regard to the case in which the bacillus subtilis was found, there was a chronic ulcer, which was probably an important factor in the causation of the thrombosis, by setting up a local phlebitis, and was, further, the probable channel by means of which the bacillus subtilis gained access to the veins.

The proteus vulgaris is found in the lungs and fæces, and cases of cystitis are described in which the organism has been isolated. The channel of infection in the case just recorded may have been the lungs or intestine, for there was no local external lesion, as in the case of the bacillus subtilis infection.

I bring forward these cases to show the importance of making bacteriological examinations, and leave the question of their exact relationship to the thrombosis an open one.

At the time this paper was in the hands of the printer, I had the opportunity of investigating another case of ante-mortem thrombosis. The thrombus was situated in the apex of the left ventricle, and as a result of embolism there were infarcts in the kidneys. I obtained the proteus vulgaris bacillus in pure culture from the thrombus and also from one of the infarcts in the kidneys. I consider this case the most important of the four,

as indicating an ante-mortem invasion of the organism. The post-mortem was performed thirteen hours and a half after death in cold weather, which makes a post-mortem invasion unlikely. The precautions taken in obtaining the cultures precluded the possibility of external contamination. The fact that the same organism was found both in the thrombus and in the infarcts is strongly suggestive of a causal relation between the organism and the morbid conditions found.

CASE 4.—Thomas P., æt. 63, was admitted into Philip ward, Guy's Hospital, under the care of Dr. Hale White, on August 13th, 1901, for difficulty in talking and swallowing. He had various indications of organic disease of his nervous system, which were thought to be of the nature of bulbar paralysis. On October 10th the second toe of the right foot was found to be showing signs of gangrene. On October 13th the third toe of the same foot was similarly affected. He died on November 11th.

The post-mortem examination was made thirteen and a half hours after death. The body was badly nourished. The brain weighed 1380 grammes. The terminal ends of both internal carotids were atheromatous, calcareous, and dilated. There was softening in both cerebral hemispheres. The branch of the left coronary artery in the anterior interventricular groove was atheromatous, calcareous, and thickened, and the lumen was practically obliterated. The apex of the left ventricle was dilated and contained a large, organised, ante-mortem thrombus which was softening in the centre, for on cutting into it a quantity of thick, sanguineo-purulent looking fluid oozed away. The lower portion of the clot was firmly adherent to the endocardium, and had a markedly laminated appearance.

The endocardium covering the portion of the ventricle which was dilated was very much thickened and was quite opaque. The corresponding part of the myocardium was very much thinned and had undergone marked fibrosis. At its thinnest part it was no thicker than the wall of a normal right auricle. There was no valvular disease.

There were several large hæmorrhagic erosions in the stomach. The large intestine was congested.

There was superficial scarring of the under surface of the right lobe of the liver.

The right kidney contained two small recent infarcts. The testes were not fibroid.

Cover glass preparations from the thrombus and one of the infarcts shewed a few short thick bacilli.

Cultures were made on agar from the thrombus and one of the infarcts. A moist, translucent, spreading growth over the surface of the agar resulted.

Gelatin plates were made, and in both cases, after twenty-four hours, at 20° C., complete liquefaction from plates 1 and 2 resulted and on plate 3, small pale, yellowish white, liquefying colonies were seen. Several tubes containing different media were inoculated from these liquefying colonies.

Cultures.

Agar.—After twenty-four hours, at a temperature of 37° C., a spreading pale, yellowish white translucent growth, which covered the surface of the gelatin, resulted.

Litmus Milk.—Was rendered slightly acid after incubation for twenty-four hours at 37° C. There was no clotting.

Urine.—Was rendered alkaline at the end of twenty-four hours at 37°, it became turbid, and there was a deposit containing crystals, which proved to be triple phosphates on microscopical examination.

Blood serum.—On this medium there was a free growth, and marked liquefaction at the end of twenty-four hours at 37° C.

Nitrate broth.—There was a good growth, and the broth became turbid. A good nitrite reaction was obtained with phenyl diamine.

Gelatin.—There was abundant gas-formation and liquefaction in shake cultures.

Microscopical examination of the cultures shewed a bacillus, which varied in size, and was motile. It stained well with methylene blue, but did not retain the stain when treated by Gram's method. It was identified as the *proteus vulgaris*.

A CASE OF SUPPURATIVE PYLEPHLEBITIS.

By FREDERICK TAYLOR, M.D.

SENIOR PHYSICIAN TO GUY'S HOSPITAL.

SUPPURATIVE PYLEPHLEBITIS is so rare that any instance of its occurrence is worthy of record, and I take the opportunity of describing the following case all the more willingly, because in a recent volume of these Reports¹ a valuable analysis is given by Dr. J. H. Bryant of twenty cases occurring at Guy's Hospital, scattered over a long period of years. The report is from the notes of Mr. F. G. Gibson.

E. W., æt. 28, was admitted under my care into Clinical ward on February 17th, 1901.

She had measles, whooping cough, and scarlet fever as a child. She has been strong since, and has suffered only some slight abdominal pains, and has taken laxatives and used a "hydrant" for constipation.

Since Christmas she has been unwell and has suffered from boils on the neck and under the chin. On the 12th inst. she had a bad headache, and pain in the abdomen; she vomited and felt "chilly." She attempted to work but had to go to bed, and had then "cramps in the stomach." On the 13th the temperature

¹ Guy's Hospital Reports, vol. liv., p. 77.

rose to 105°, and she had more shivering, involving the whole body, and this occurred four times. The fever and headache continued until her admission.

On admission.—A well-developed and well-nourished woman, milliner by occupation and unmarried. She is excited rather than drowsy and has some pain in the abdomen, which she does not localise.

The tongue is furred. The abdomen is full, moves well on respiration, and presents a few doubtful rose spots on the left side. The margin of the liver can be just felt below the costal margin; it is not tender even to deep pressure. The spleen can be felt projecting, on deep inspiration, an inch and a half into the abdomen, and the splenic dulness is correspondingly increased. In the left flank, close to the spine, the kidney can be felt on bimanual examination. In the right iliac fossa there is marked gurgling on pressure. No peritoneal rub can be heard over liver or spleen.

The motions are of the colour and consistency of pea soup, and are not offensive in odour.

The chest appears normal on auscultation and percussion, with the exception of a few sibilant rhonchi in front at the upper part. There is also slight cough, but no expectoration.

The heart is normal; pulse 124, regular and full. A blood-count gives red corpuscles 4,670,000, leucocytes 10,500, hæmoglobin 56 per cent. The reflexes and special senses are normal.

The urine is of specific gravity 1020, acid in reaction, depositing urates abundantly, and containing a trace of albumin, but no blood, pus, or sugar.

The menses are generally regular, but on this occasion commenced ten days before the expected time.

Progress of case.—She had very much the appearance of a patient with typhoid fever, and on February 21st the serum gave Widal's reaction, but on the 22nd she had two rigors, followed by profuse sweating. On the 24th three rigors; no signs in the chest; the spleen scarcely palpable; bowels opened at mid-day; patient not drowsy; feels comfortable except during the rigors. On the 25th three rigors, followed by vomiting; a small amount of

blood was passed with the stools. On the 26th, epigastric pains; no signs in chest; no evidence of a source of pyæmia in bones or limbs; the heart-sounds clear. Pulse 100 to 120, regular.

February 28th. Has occasional rigors, during which the pulse becomes weaker, and the face dusky and pinched, the left side more than the right.

March 7th. During the week there have been rigors as follows: one on March 1st, three on the 2nd, one on the 5th, and two to-day. The motions have been frequent, on some days as many as five, very fluid, olive green in colour, not offensive, and containing no sloughs, but on the 5th a little blood. No cardiac bruits, no adventitious sounds in chest. Spleen felt on deep inspiration.

March 12th. Patient much thinner. Diarrhœa has been profuse, and on the 9th there was rather severe hæmorrhage. A Widal reaction on the 8th was negative. To-day there is pain in the legs and thighs, the calves and pads of the toes on the right foot are tender; knee-jerks depressed. At 1 a.m. she retched violently, and fell back in a faint, became dusky and almost pulseless. The condition of collapse continued in spite of stimulants and transfusion of saline solution, and she died at 6 a.m.

The treatment consisted of milk diet; salol internally; on the 24th, quinine in two-grain doses. After this, opium enemata were used to check the diarrhœa, and brandy was administered freely towards the end. The temperature, taken every four hours, ranged irregularly between 98° and 104°, rising and falling often more than once daily, without any reference to morning or evening hours. It often reached 105° in the rigors, of which there were sixteen recorded, and once 106°. The pulse was always 100 or more, and at some part of every day was 120 or 130. The respirations were commonly from 24 to 32.

Post-mortem.—The inspection was made by Dr. Fawcett on March 12th. On opening the abdomen, the omentum was found to be adherent to the underlying coils of small intestine, and these coils were united together by recent deposit of lymph. On separating these coils a small abscess cavity was opened in the

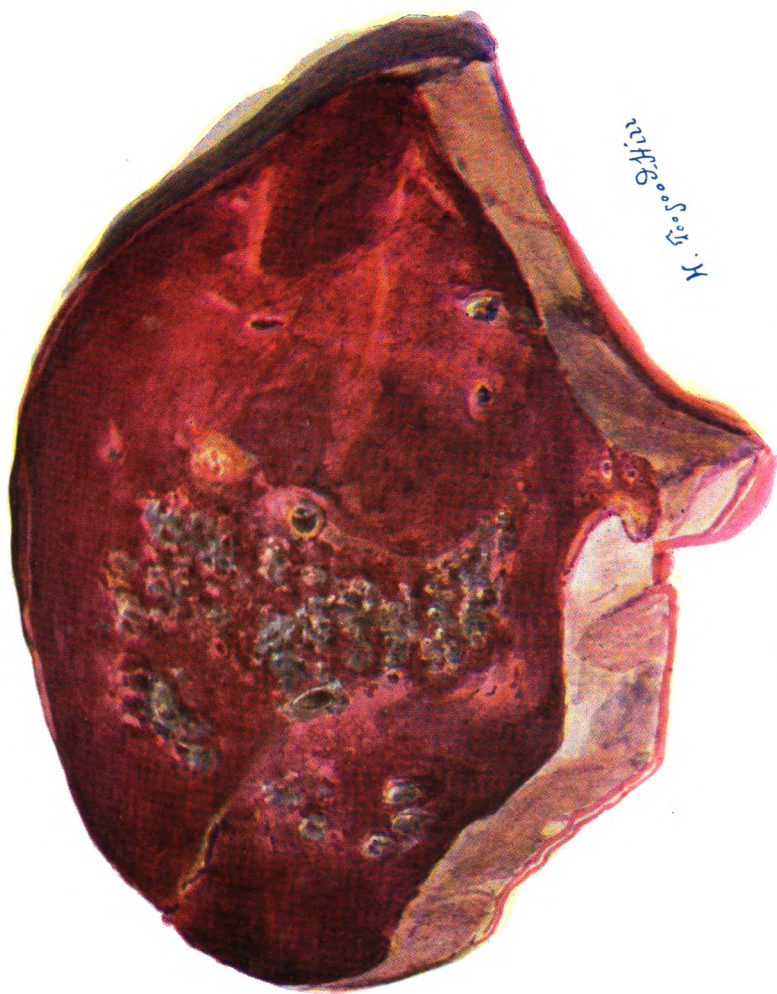
right iliac region internal to the lower half of the ascending colon. The cavity was shut off by adhesions from the general peritoneal cavity, and its walls were formed on the inner side and in front by coils of small intestine, and above by the hepatic flexure of the colon, which had been pulled down over it. Opening into the cavity was a hole which led into a thickened and ulcerated appendix cæci. The appendix came off from the inner side, and was running upwards along the inner aspect of the ascending colon, and then inwards.

At the root of the appendix a gangrenous process had supervened, and the sloughing base of it was found to open into a ragged cavity measuring 7 cm. by 4 cm. situated behind the ascending colon. The walls of this cavity were of a dirty black-grey colour, and at the upper extremity there were several small round openings into the ascending colon, at a distance of 6 cm. above the ileo-cæcal valve. The edges of these openings were acutely inflamed.

At a position corresponding to the origin of the appendix was the mucous membrane, of deep red colour, with grey membranous patches upon it. The attached portion of the mesentery was much thickened, and its veins were full of a blackish-grey, purulent fluid. The other main branches of the portal system were also affected in the same manner, the splenic vein and main trunk of the portal vein being lined by a yellow membrane very similar to that known as the pyogenic membrane of an ordinary abscess. Apart from the above changes, the intestines, small and large, were healthy.

The *liver* weighed 1432 grammes; it was dark red in colour for the most part, with a few scattered, yellow areas varying in size, which on section yielded pus. On section of the organ, numerous abscesses varying in size from that of a pin's head to that of a pea or threepenny-piece, were seen scattered throughout. (*see plate.*) The portal vein branches were full of blackish-grey, purulent material. The hepatic veins and biliary ducts were normal.

A Case of Suppurative Pylephlebitis.



A section of the liver, showing a large number of small abscesses.

The *spleen* weighed 150 grammes, and contained an infarct. The other organs were normal: the heart weighed 231 grammes and the kidneys 250 grammes.

Bacteriological report.—Pus from abscesses in the liver, and blood from the heart, taken eight hours after death. The blood from the auricle contained a pure culture of *bacillus coli communis*. The pus from the abscess in the liver also gave a pure culture of *bacillus coli communis*.

The above is a good example of suppurative pylephlebitis, and so far as the pathological and ætiological conditions are concerned it resembles many of the cases already recorded. Suppurative pylephlebitis is, as the name implies, an inflammation of the portal vein resulting in the formation of pus. It is due, practically in every case, either to a lesion of some structure within the tributary area of the portal vein, or exceptionally to a lesion of the liver itself, involving the hepatic distribution of the vein and not of the trunk and tributaries. As a result of such lesions, in the former case, septic organisms are conveyed into the portal vein, thrombosis and septic invasion of the clot take place, and from this again septic particles are conveyed in the portal vein distributed throughout the liver so that numbers of minute abscesses in this organ are the result. In the latter case the disease begins in the hepatic branches of the portal vein, and if these are moderately large the same dissemination of septic particles through the smaller veins can take place as in cases where the causative lesion is on the tributary side of the vein.

Every part of the portal area may be the seat of the lesion which is the primary cause of suppurative pylephlebitis. In the twenty cases collected by Dr. Bryant from the Guy's Hospital records, the causes were ulcers of the stomach, duodenum, colon, or rectum, appendicitis, gall-stones, and suppurating gall-bladder, pyosalpinx, and suppurating ovary, and abortion. From other writers he notes in addition, sloughing of the cæcum, suppuration in the spleen or mesenteric glands or about the hæmorrhoidal

veins, inflammation of the umbilical vein in infants, and the implication of the mesenteric vein by a fish-bone.

Chvostek, in a very complete article,² published some years ago, besides most of the above, mentions also suppurative pancreatitis, abscess between the layers of the mesentery, localised peritonitis from a fall, and hepatic abscess or hydatid cyst, involving the hepatic branches of the portal vein.

It is thus seen that no part of the abdomen, which drains its blood into the portal area, is exempt from the liability to cause suppurative pylephlebitis. The most frequent lesion is probably appendicitis. Of the twenty cases recorded from Guy's, it was the cause in eight, and the present case adds another. Chvostek also mentions appendicitis as the most frequent cause.

Dr. Bryant calls attention to one notable exemption, that is, the absence among the Guy's cases of any in which typhoid ulceration was the cause. From this alone, seeing that the number of cases is so small, perhaps not much can be said; but no doubt, as he remarks, typhoid ulceration is the most common form in this country. Chvostek also, does not appear to have found typhoid as a cause. The explanation of this common exemption may be found in the bacteriology of the disease. Streptococci, staphylococci, and the bacillus coli communis have been most often found; the latter was present in my case. The typhoid bacillus may have less facility for transmission in this particular way; and yet it has been found in the pus of abscesses resulting from periostitis after typhoid fever, and more to the point, in the gall-bladder in typhoid cholecystitis. I should still think it likely that cases will be, or may have been already reported; but even then the occurrence must be very rare, as compared with the position which appendicitis holds.

I now come to the question of symptoms and to their recognition clinically in different cases, *i.e.*, to the diagnosis. Briefly stated, the condition is one of pyæmia, with more or less evidence of abdominal disease, which may be of two kinds, one the

² *Klinische Vorträge über die Krankheiten der Pfortader und der Lebervenen. Wiener Klinik, 1882, pp. 67-106.*

original suppurative or ulcerating lesion, the other, the changes in the liver which result therefrom.

In a typical case, therefore, one might expect to get, first, the signs of the original local disease, whether it be appendicitis, gastric ulcer, or pelvic inflammation, with such local pain, tenderness, and resistance to pressure as they may cause.

Secondly, the development of a definitely pyæmic condition as shown by rigors, great oscillations of temperature, undue prostration, drowsiness, sallow tint of complexion, etc., and thirdly, the evidences of hepatic disease, namely :—

- (α) Pain, tenderness, enlargement of the liver.
- (β) Jaundice from pressure on the bile-ducts.
- (γ) Enlargement of the spleen, diarrhœa, and sometimes hæmorrhage from pressure on the portal vein.
- (δ) Possible extension of inflammation through the diaphragm resulting in a right-sided pleurisy.
- (ε) Possibly peritonitis.

Towards the end the typhoid or pyæmic condition becomes the prominent feature; the patient is emaciated, sallow, drowsy, or semi-comatose, occasionally delirious, with rapid, feeble pulse. The diagnosis is admittedly difficult, and in a great number of cases is not made during life. By a careful consideration of the symptoms, and of their relative frequency, we shall see how this is so.

Chvostek mentions the following diseases as likely to be confounded with suppurative pylephlebitis: pyæmia, septicæmia, typhoid fever, malaria, adhesive pylephlebitis, catarrhal and other forms of jaundice, chronic catarrh of the bile-ducts, biliary calculus and cirrhosis of the liver. Dr. Bryant points out that his colleagues and predecessors at Guy's have succeeded in adding to that list catarrhal pneumonia, general septic pneumonia, empyema, subdiaphragmatic abscess, spinal caries, and rupture of a reduced hernia.

The frequency with which among the Guy's Hospital cases a pulmonary or thoracic lesion was regarded as the primary disease, emphasises the fact that the hepatic inflammation tends to spread to the chest, a fact to which Chvostek makes but little reference.

At the same time it suggests one great difficulty in the diagnosis of this disease, namely, the frequent latency or slight clinical importance of the primary abdominal lesion. It will be convenient to take seriatim the several features of the disease which Chvostek regards as guides for diagnosis and compare them with the facts of my case and of others.

1. *Demonstration of a possible cause.*—It is a familiar fact that such abdominal lesions as are enumerated above, are either entirely latent, or so little distinctive in their symptoms, as to make their recognition a matter of great uncertainty. Appendicitis, which is a very common cause of suppurative pylephlebitis, may in its gangrenous form be entirely latent until an acute and probably fatal peritonitis is set up. In the present case the illness commenced, according to the patient, with abdominal pain, vomiting and shivering five days before admission; on the following day she had four rigors, and on admission the spleen was already enlarged. The only sign here, which could be referred to appendicitis, was some gurgling in the right iliac fossa; and this was obviously more suggestive of typhoid fever than of appendicitis. Not that I have much confidence in the value of this sign as a diagnostic feature in typhoid fever, and there is no doubt that a large proportion of typhoid cases do not show it. But, on the other hand, it is not a characteristic feature of appendicitis, in which a more or less firm resistance, with tenderness and localised dulness are the more common signs. What the conditions of the right iliac fossa may have been prior to admission I have no means of knowing, but it seems to me probable that the recognised commencement of the illness was determined by the pyæmic infection, and not by the gangrenous change in the appendix. This is, at any rate, in accordance with the experience of the cases already referred to, in which the symptoms fatal in two or three days are referable entirely to a general acute peritonitis, and little, if at all, to the gangrene of the appendix, which must have preceded it.

2. *Exclusion of causes of pyæmia and septicæmia in the systemic circulation and of ulcerative endocarditis; and*

3. *The existence of septic fever.*

These may be taken together, but in reversed order.

There is, of course, the old difficulty of the specific distinctions of the various forms of pyrexia. The septic, or septicæmic, or pyæmic character of the fever must be shown before a diagnosis of such a rare complaint as this can be made. Every one of the primary lesions, which may be the cause of it, is commonly accompanied by some degree of pyrexia. If such a lesion is recognised by local evidence, pyrexia in some form is to be expected, and there is no need to be alarmed as to the existence of pylephlebitis. If a local lesion is not recognised, then for some days possibly one will be led to discuss the toxic conditions or infectious fevers, which may exist with a minimum or an entire absence of local signs, such as typhoid fever, tuberculosis, or influenza. Typhoid fever is extremely likely to be suggested from its frequency, from the great variety in the mode of its occurrence, and from the large proportion of cases in which local evidence of the disease in Peyer's patches is early wanting; and in many cases a diagnosis of this kind has been for a time made and entertained. In the present case there were four points in favour of typhoid fever on her admission on the sixth day of the illness, namely, headache, some spots on the abdomen admittedly not very convincing, enlargement of the spleen, and the gurgling in the right iliac fossa before referred to. In addition to this we had diarrhoea and frequent motions after the fourteenth day, a successful Widal reaction on the tenth day, and hæmorrhage from the bowels on the fourteenth, twenty-second and twenty-sixth days of the illness, the last severe. But the chief feature against typhoid fever was the early appearance and the frequent occurrence of rigors; and these combined with the sallow, earthy, and septic appearance of the patient in the latter part of her illness, determined the exclusion of the diagnosis of typhoid fever, and the recognition of the fact that she was suffering from one or another form of pyæmia.

Not, however, that rigors of themselves are unknown in typhoid fever; they are very uncommon, but it is well known that they occur. Two years ago I devoted some part of a clinical lecture to rigors in typhoid fever, based on a case under my care,⁵ and

⁵ *Guy's Hospital Gazette*, June 23rd, 1900.

I was able to refer to other cases that I had seen. Professor Osler also has written on this subject in one of his reports from Johns Hopkins Hospital,⁴ and in his series of two hundred and twenty-nine cases of typhoid fever, there were thirteen in which rigors occurred. Constipation and acute local inflammation, like pneumonia, are sometimes the cause, but occasionally they occur for no obvious reason, and cease, without prejudicing the patient in any way.

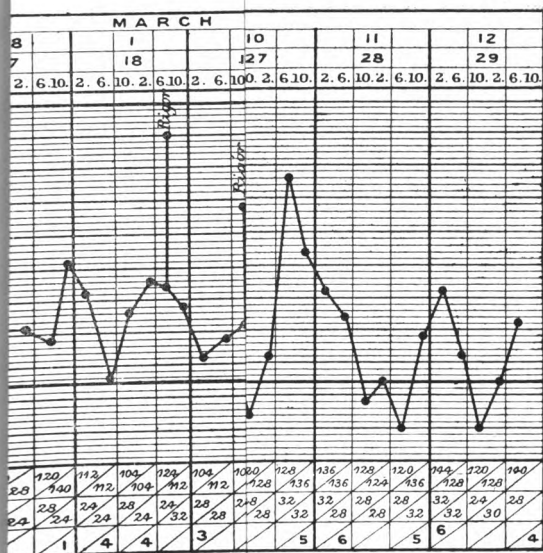
Another feature in the case opposed to typhoid fever was the course of the temperature (see fig. 1), when we had sufficient of it to know the general course it was taking. I need not discuss the well-known features of a typical typhoid chart, nor the frequency with which the course of temperature in typhoid is not typical.

We come, then, to recognise that this patient was pyæmic, and to inquire for the source of the pyæmia. Chvostek rightly sees that a process of exclusion may bring us to the diagnosis. If the systemic circulation on the venous side (ordinary pyæmia), and the heart or cardiac valves (malignant endocarditis), can be excluded, then a portal pyæmia alone is left. Unfortunately, experience tells us that it is very difficult to exclude malignant endocarditis and it is well known that this form of pyæmia (arterial pyæmia) may exist without any indication, by murmur or by irregular action, or by altered position or impulse, that the heart is the seat of such a serious and fatal lesion. Instances could be easily multiplied of this occurrence, and the following case, which occurred a few years ago under my care, is an exceedingly good example. It is all the more interesting here, because the medical man who sent her into the hospital believed she had appendicitis; and had that view been maintained for any length of time after her admission, we ought, proceeding on the lines of Chvostek's formulæ, to have further diagnosed the occurrence of suppurative pylephlebitis.

The patient was a woman aged 50. She had been for years subject to pains in the limbs; four and a half years previously pain over the region of the liver for a fortnight; constipated for

⁴ The Johns Hopkins Hospital Reports, vol. v., p. 445.

urative Pylephlebitis



G. 1.

104.2°, in the second 102.8°. In the intervals it never fell below 99°. On the 18th she had much pain in the right side, but was

I was able to refer to other cases of this kind.

kins Hospital Reports, vol. v., p. 445.

the last twelve months; never jaundiced. For three or four weeks she has had a cold and has felt unwell. On December 10th she woke up with a pain in the abdomen and headache. She vomited three times a slimy fluid with no blood. She seems to have improved somewhat, but the pain was continuous, dull, sharper at intervals, and running through to the back. On the night of December 15th she had a rigor, and on the following morning she vomited green fluid.

The doctor who first saw her on the 10th, wrote: "Her symptoms were subacute, and the fever subsided quickly; the temperature was normal on the 13th and 14th, but lighted up again suddenly on the night of the 15th; she looks very ill." He regarded it as a case of acute appendicitis and sent her to the hospital on December 16th.

She appeared well nourished, and her cheeks were of good colour. She lay in bed, apparently in no great pain, but with some shortness of breath. The pulse was 108, the respirations 32, and the temperature 99°.

The abdomen was not distended, and it moved with respiration. There was no visible peristalsis, and there were no spots. On deep palpation, resistance was met with at the junction of the epigastric and right hypochondriac regions, as well as in the neighbourhood of the right kidney, and between the umbilicus and the right iliac spine, about one inch from the latter. There was also a little tenderness near the right eighth rib and the right iliac spine. Nothing could be definitely felt. The liver was not enlarged, nor was the spleen.

The impulse of the heart was normal, the heart-sounds were faint, but no adventitious sounds were heard. The lungs appeared to be healthy. The urine was light yellow in colour, deposited urates on cooling, had a specific gravity of 1028, and was free from sugar and albumin.

In the evening the temperature rose to 102°. On December 17th she had a rigor, the temperature rising to 101·6°. On the 19th she had two rigors, the temperature in the first reaching 104·2°, in the second 102·8°. In the intervals it never fell below 99°. On the 18th she had much pain in the right side, but was

free from it on the 19th. She had rigors again on the 21st and 22nd.

The sclerotics were thought to be slightly yellow on the 21st, but they were not so on the 23rd. On this last day the right iliac fossa was quite supple, and the fingers could be pressed deeply into it, without discomfort. The temperature on this day was between 100.4° and 102.6° , and on the 24th occurred another rigor.

The temperature reached 103° on the 25th, and gradually fell during the next six days to 100° – 101° , after which it rose a little, keeping an irregular course between 99° and 102° , with no very extensive oscillations.

On December 28th there was a trace of albumin in the urine; the patient was flushed, but not jaundiced, and nothing could be felt in the abdomen. An examination of the pelvic organs on the 24th revealed nothing abnormal. The bowels had been moved, on an average, once a day, and there was nothing remarkable in the motions.

On January 2nd, sibilant rhonchi were heard in the chest, back and front; sonorous rhonchi towards the axillæ; and mucous râles in the second and third intercostal spaces.

On January 4th, she was slightly jaundiced, and she vomited undigested food and bile. Rhonchi as before, crackling râles at the bases behind, and some impairment of resonance over the middle of the right lower lobe. She complained of no pain in the abdomen, but in the shoulders and hips; also of prostration and shortness of breath. The liver was felt two inches below the right costal margin, and the spleen extended below the left ribs, moving freely with respiration. On the following day the crackling râles extended to the axilla, and an area of broncophony was found over the right scapula. The next few days there was increasing distress, a sense of dyspnœa, twitching of the *alæ nasi*, dusky flush in the face, tremors of the hands, frequent retching and occasional vomiting. The signs in the chest persisted; nothing wrong was discovered over the heart. The albumin found in the urine again disappeared, and the jaundice subsided.

On January 10th and 11th, the temperature was mainly below 101°; it ranged between 97° and 99·8° on the 12th. She was now rapidly sinking, and she died on the 13th, the temperature being 97·8°. The pulse throughout had varied from 110 to 120, the respirations from 26 to 36.

Post-mortem.—Septic endocarditis of the mitral valve was found, with pneumonic foci, and a hæmorrhage in the right lung, a large soft spleen, kidneys scarred by infarcts, and a small renal calculus on the right side.

4. *The evidence of suppurative hepatitis, e.g., local pain, swelling, and especially the elevations due to abscesses.* In my case the insignificance of any local signs of disease of the liver was very interesting. *Hepatic pain* was certainly not a striking feature in her case. On admission she had abdominal pain, which she could not localise; later on she was entirely comfortable but for the rigors. On another occasion there was epigastric pain, which may, of course, have been due to the liver. *Hepatic swelling* scarcely existed, certainly not to an extent which would compel one to consider a suppurative hepatitis the most probable condition. On admission the “lower margin of the liver was just felt below the costal margin; it was *not tender on deep palpation.*” The abdomen was, of course, frequently palpated afterwards, and no serious enlargement was observed. Moreover, after death the liver weighed only 1,432 grammes, that is, 50·5 ounces, surely not a great size for the liver of a medium-sized female. Still the fact remains that the liver is often enlarged somewhat in these cases, and a frequent observation in the cases collected by Dr. Bryant is that the liver can be felt just beneath the costal margin; but when the enlargement is so little the value of the change must depend upon the association with other symptoms, such as pain, tenderness and jaundice.

The prominences on the surface of the liver, to which Chvostek refers, appears to be relatively infrequent.

It is important to note further that jaundice, which would necessarily attract attention to the liver, is by no means necessarily present. This is well recognised, but the proportion of cases in which it is present is given differently by observers,

working in all instances on small collections of cases. Chvostek says it is common, and quotes Frerichs as finding in it 75 per cent. of recorded cases, whilst Chvostek's own cases gave six out of seven, or 85 per cent. The Guy's cases showed it in only 40 per cent., and in 55 per cent. it was expressly stated to be absent. The present case had no jaundice, and it is clear that one must try to make a diagnosis without this factor.

5. *Evidences of stagnation of blood in the portal vein district.*—Of the five signs under this head mentioned by Chvostek, three are admitted by him to be rare, namely, *hæmatemesis*; *ascites* (rarer) and *distension of the abdominal veins* (rarest of all). The remaining two signs are *enlargement of the spleen* and *diarrhœa*. They were both present in this case, and they are clearly not infrequent. But unfortunately they are not distinctive, and they are even misleading, since they are two out of the most trusted witnesses to typhoid fever, a disease with which suppurative phlebitis is very likely on other grounds to be confounded.

I know of no means by which the enlarged spleen of one of these diseases can be distinguished from that of the other, unless it be by the bacteriological examination of blood drawn from the spleen for Eberth's bacillus, a process which Dreschfeld⁵ says cannot be countenanced for this purpose.

With regard to the stools, it is certain that in spite of the full descriptions of what we are all inclined to regard as the distinctive features of typhoid stools, their ochrey yellow colour, pea-soupy appearance, the presence of sloughs, the alkaline reaction, "offensive" odour, or rather peculiar odour to those who can distinguish it (for no stools can be called fragrant), it is doubtful if any physician would stake his reputation on an absolute diagnosis by the stools alone in every case. It is quite certain that the physical characters of typhoid stools are very closely imitated by those in other diseases, of which I may mention tubercular disease of the bowels and ulcerative colitis; but it is also certain that the stools in typhoid fever, even when diarrhœa is present, are not always ochrey, or yellow in colour, but may be green or blackish-green. Here again bacteriology is suggested. Dresch-

⁵ Allbutt's System of Medicine, vol. i., p. 835.

feld⁶ says with regard to the detection of typhoid bacilli in the stools that their "isolation and distinction from the bacterium coli take much time, and are processes too elaborate to be clinically useful; moreover, the bacilli have not been found in the stool before the eighth or ninth day of the fever."

As four years have elapsed since this opinion was published, I have asked our bacteriologist, Mr. W. C. C. Pakes,⁷ to let me know the present state of the question. He writes thus:—"Although I have succeeded in isolating the bacillus a few times, I have failed many times, and I have certainly come to the conclusion that the labour is so excessive in comparison with the paucity of results that it is only in the really important cases that it is worth while trying." And he says: "It is all but impossible to isolate the *B. typhosus* from fæces in which this organism is in great numerical inferiority." He agrees that they are unlikely to be found before the eighth or ninth day, as they are probably only yielded by the ulcerated surface.

He goes on to say: "The cases, in which I think that the investigation would be useful, are those that are thought to be enteric; but which remain doubtful clinically, or fail to give Widal's reaction. The isolation of the *B. typhosus* from the stools would, in my opinion, clear up the diagnosis, the failure to find them would, of course, leave the physician *in statu quo*."

The other three symptoms under the head of portal stagnation, namely, hæmatemesis, ascites, and distension of veins, are all of rare occurrence. The first two would certainly direct attention to the liver, and even to the portal vein, without having pathognomonic value. The third might suggest compression of the inferior vena cava, and thus only localise the disease deep in the abdomen, without further distinctive assistance.

6. *Local pain*.—The value of this from a diagnostic point of view is probably not very great. First, the pain may be very insignificant, and secondly, the relation of pain in particular situations to the organs supposed to correspond to these situations is known

⁶ Loc. cit. p. 835.

⁷ Since this was written, Mr. Pakes has been appointed Bacteriologist and Public Analyst to the Transvaal Colony.

by experience to be variable. From a given pain in the right side, appendicitis, gall-stone, and renal calculus may all be diagnosed by different physicians or surgeons; and pain in the right flank, which is believed to be hepatic, may prove to result from pleurisy or pneumonia. It is very doubtful whether there is any material difference between the pain which may result from pleurisy, hepatic congestion, and distension of the colon. I am quite sure that the pains of gastric distension, and of post-herpetic neuritis may be indistinguishable to the patient. A definite tenderness over the liver is probably of much more value, and combined with enlargement of that organ, should draw sufficient attention to the possibility of hepatic inflammation to bring one very near to a diagnosis. In the present case it is expressly stated that the liver was not tender, even on deep pressure. The pain was sometimes "not localised," at another time it was "in the epigastrium."

7. *Prostration and Emaciation.*

8. *Peritonitis.*

Obviously these have no diagnostic value. The first are the results of any sufficiently prolonged and severe infectious disease. Peritonitis is a common result of any similar infection localised in an abdominal organ.

The signs and symptoms selected by Dr. Bryant⁸ as valuable in the diagnosis of this disease may be seen to cover the same ground as those of Chvostek, though more briefly stated. "Pyrexia, rigors, sweating, rapid pulse, abdominal pain, and tenderness in the right hypochondriac or the epigastric region, together with uniform enlargement of the liver, and tenderness of that organ, especially if associated with or following any ulcerative lesion of the alimentary tract below the œsophagus, should suggest the possibility of suppurative pylephlebitis."

That is practically the recognition of—

- (a). A condition of pyæmia.
- (b). A source of infection in the abdomen; and
- (c). The implication of the liver.

⁸ *Loc. cit.*, p. 88.

But I have already shown how it may be difficult to establish or easy to overlook the evidence with regard both to (b) and to (c), and thus we may be face to face with a simple pyæmia. If no cause can be found for this in the systemic circulation, and the heart is shewn to be normal, then the abdominal or portal circulation is alone left to supply the septic focus for the pyæmic condition.

This formula is seen to be practically the same as a statement of Chvostek's second and third points (*see* p. 116) in inverse order, namely, the existence of pyæmia or septic fever which is due neither to a lesion in the systemic circulation, nor to malignant endocarditis. Confirmation would then come from such evidence as could be found in the abdomen of the primary septic focus, and of secondary implication of the liver.

Even this may fail us in every one of its three divisions, for first, it is known that the only evidence of malignant endocarditis may be a cardiac murmur, and that a murmur is sometimes wanting. Secondly, it is conceivable that a minute lesion in the general circulation may be overlooked, though sufficient to serve as a focus of pyæmic infection. Thirdly, that the septic or pyæmic nature of an infection in the sense here meant cannot be safely inferred without the occurrence of rigors. Now, rigors are not constant in suppurative phlebitis. Chvostek, it is true, speaks of them almost as if they were. He says,⁹ "Fever is constantly observed in this process, and it is quite analogous to that which is observed in pyæmia. Attacks of shivering occur followed by heat and profuse sweating, of which the first according to Frerichs, should indicate the formation of pus in the portal vein." He then points out that the attacks are not regular in occurrence, but from their commencement quite irregular, sometimes at intervals of two or more days, at other times occurring twice or even four times in the day. Later on he says, "In protracted cases it became a remittent fever with morning remissions and severe exacerbations in the evening, and this usually passes, with increasing weakness of the patient, into an intermittent

⁹ Loc. cit., p. 94.

fever, in which the attacks do not begin with shivering, but are nevertheless terminated by profuse night sweats."

In the Guy's cases, however, Dr. Bryant found rigors were noted in only 50 per cent. of the cases, and were stated not to have occurred in 40 per cent. Some of these may have been in the late prostrate condition referred to by Chvostek.

The fact remains, however, that rigors, which are regarded as the distinctive feature of pyæmic infection, are not always present.

It may be said that few diseases have pathogenic features by which we can recognise them, and that in most diseases an analysis such as the above would show that there was nothing to be absolutely relied upon, and yet they are frequently, nay constantly, diagnosed with success. There is, of course, one factor in diagnosis which is of great importance at all times, and that is our acquaintance with the disease, and our knowledge of its probability. Suppurative pylephlebitis is very rare. Dr. Bryant shows that the post-mortem records of Guy's Hospital contain only eleven cases in twenty years. Under these circumstances the probability of suppurative pylephlebitis being present in any given case of septic disease is very small, and even the possibility of its occurrence may easily be overlooked.

Malignant endocarditis was, no doubt, at one time in the same position. It is desirable when we have to do with an obscure septic condition to remember that the cause may be in the portal as well as in the systemic circulation. The chief aid to the diagnosis of the disease is the knowledge that it may occur.

BLUE URINE.

By A. P. BEDDARD, M.D.

SENIOR DEMONSTRATOR OF PHYSIOLOGY.

It is well known that when methylene-blue is taken by the mouth it reappears in the urine, giving it a deep green or peacock-blue colour, according to the dose. Occasionally, however, a green or blue urine is passed by a patient who is not taking medicine in any form, and whose occupation does not suggest any possible means of obtaining methylene-blue. In such a case it becomes necessary to determine the nature of the pigment.

Morley Fletcher (Clin. Soc. Trans., vol. xxxii.), reported two cases of an apparently spontaneous passage of methylene-blue in the urine, and the following is a similar case. A country labourer, aged 30, came up to the out-patient department, complaining that for several days he had been passing more or less discoloured urine. He had taken no medicine nor pills of any kind, had nothing to do with paints or dyes, and had neither sucked nor swallowed any dyed material. He passed a specimen of urine with the following characters:—It was of a rich peacock-blue colour, distinctly acid in reaction, and normal in all other respects. The urine was placed in a bottle and corked, and on the next day it was found that the blue colour had entirely disappeared, leaving the urine of an ordinary yellow colour. On shaking it up with air, however, the blue colour rapidly returned, and after the addition of a few drops of formalin, has never again disappeared.

The power of living tissues to remove oxygen from methylene-blue and convert it into a colourless compound is well known, and in this case was exerted by a growth of bacteria in the urine. In passing, attention may be drawn to the remarkable fact that methylene-blue is apparently able to exist in the urine in contact with the tissues of the bladder in its blue condition, and this is all the more remarkable in that urine contains the merest traces of oxygen.

When this urine was examined, it could be seen that the blue pigment was present, partly in solution and partly as blue amorphous granules, and when filtered a blue deposit was left on the paper, the filtrate being not blue, but green. The blue deposit was soluble in water, producing a clear blue solution.

Green urine may be caused by the presence in the urine of biliverdin, by carboluria, or by indigo-blue or methylene-blue taken by the mouth. The first two can be readily recognised by the ordinary tests, it only remains to distinguish between the two latter.

Again, blue urine may be due to methylene-blue or indigo-blue. So far as is known, methylene-blue cannot be manufactured in the body, but must have been absorbed as such. Indigo-blue may be either absorbed as such or derived from proteid decomposition in the alimentary canal. In the latter case, usually not indigo-blue itself, but its colourless chromogens indican or indoxyl glycuronate are passed. More rarely, however, indigo-blue may be present in the urine when passed; its presence is due to an alkaline fermentation taking place in the renal passages and splitting off from the chromogens the indoxyl, which becomes oxidised into indigo-blue. Consequently, indigo-blue, unless it has been absorbed, is only present as such in alkaline urines, although it has been recorded in one instance in an acid urine.

To distinguish between indigo-blue and methylene-blue in urine it is only necessary to examine the urine spectroscopically, for the characteristic bands of the two substances are in such widely different parts of the spectrum that the diagnosis can be made with certainty with an ordinary pocket spectroscope. Both pigments are extracted from urine by chloroform, and when present

in the urine in only small quantities it is well to examine a chloroform extract. Indigo-blue gives a broad band lying on the yellow sodium line D. Methylene-blue, on the other hand, gives a much narrower and darker band in the extreme red end of the field of an ordinary pocket spectroscope.

If a spectroscope be not available, the diagnosis is still possible by noting the following points :—

(1). The reaction of the urine. If a blue urine is acid when passed, it is probably not coloured by indigo-blue. If alkaline, the colour may be due to either substance.

(2). Extraction with ether. Although chloroform extracts both of the pigments from urine, ether only takes up indigo-blue.

(3). Disappearance of the blue colour in a corked bottle and reappearance on shaking with air. Methylene-blue in urine may be reduced by the growth of bacteria in a single night to a colourless compound. Indigo-blue may also be reduced to indigo-white by bacteria, but the conversion takes much longer. Both blue colours rapidly reappear on shaking the urine with air.

(4). Heating two specimens of the blue urine with strong mineral acids, one with HCl, the other with HNO₃, and noting their behaviour. With HCl the blue colour of methylene-blue rapidly disappears, and if the urine be then shaken vigorously with air, its blue or green colour reappears. The colour of indigo-blue, on the contrary, is not discharged.

With HNO₃ exactly the opposite is true; indigo-blue is split up into isatin, its blue colour is destroyed and will not reappear on shaking with air. Methylene-blue, on the other hand, is unaltered.

These colour changes are, however, complicated by the fact that urine frequently contains two chromogens, which, the heating with mineral acids, converts into red pigments, namely urorosein and so-called indigo-red. Either may be present alone, or both together. Urorosein may be distinguished from indigo-red by the following reactions—it is taken up from its solutions by amyl alcohol and not by chloroform or ether, it is rapidly decolorised by alkalies, and gives a broad absorption band midway between D and E.

It thus comes about that heating a blue urine with HCl in the case of methylene-blue, may turn it pink or red, and in the case of indigo-blue a mixture of red with the blue. Heating with HNO_3 may turn an indigo-blue urine red, and a methylene-blue urine a mixture of green or blue with red.

In the *Lancet* of June 1st, 1901, the case is recorded of a man who on several occasions, for no known reason, passed either peacock-blue or sage-green urine, which was acid and otherwise normal. When corked the blue colour disappeared and returned on shaking with air. The blue or green colour was considered to be due to indigo-blue, because when the urine was heated with HCl it turned pink, which was held to prove the presence of indican, and therefore of the source of the indigo-blue. From what has been said above, however, it may be concluded that this case of blue urine was one of urine containing not preformed indigo-blue, but methylene-blue absorbed from some unknown source.

REDUCTION EN MASSE.

BY R. P. ROWLANDS, F.R.C.S.

DEMONSTRATOR OF ANATOMY, GUY'S HOSPITAL.

It is not common for a strangulated hernia to be reduced en masse; during the last forty years only fifteen instances have occurred at Guy's Hospital. It is, however, an unfortunate and grave accident that may be produced by anyone who tries to reduce a strangulated hernia. It is an injury that should rarely be inflicted by the medical man, yet it is he who usually inflicts it. When it has occurred, it requires very prompt recognition and treatment. There is so little said about it in the ordinary surgical works that I have ventured to write this paper, which is based upon one hundred cases collected from various sources. I am much indebted to the surgeons of Guy's Hospital for allowing me to make use of their cases. I have arranged the cases in a tabular form, adopting the method employed by Mr. Birkett in his paper. My own case I have reported in full because of some interesting features which it presented.

Reduction en masse is commonest in cases of inguinal hernia, it is much less common in femoral hernia, and extremely rare in all other forms of hernia.

Two very doubtful cases of reduction en masse of ventral hernia are mentioned by Blackman.

Saviard¹ was the first to draw the attention of the profession to this interesting accident. He witnessed an operation at which the surgeon, mistaking the sac for intestine, reduced it. At the autopsy, Saviard found the sac lying in the subperitoneal tissue and enclosing strangulated bowel.

Le Dran, thirty years later, published an interesting case. The French surgeons of the time, however, were not convinced of the possibility of the accident, and severely criticised Le Dran's account of it.

Sir Charles Bell² was the first English surgeon to publish a case.

Luke³ gave a systematic account of the condition in 1850.

Birkett⁴ contributed a most valuable paper on the subject in 1859. Mr. Birkett collected and tabulated 37 cases, which have been included in the tables which follow these observations.

Blackman⁵ also analysed the cases recorded before 1851.

In reduction en masse the hernial tumour has been reduced from its usual position, but the intestine is still strangulated by the neck of the sac. This definition is perhaps too comprehensive, but it is founded on a clinical basis. Literally, the term should be applied to only the first of the following varieties. Common and long extension of the name to the other varieties, and especially the clinical similarity of these justify their inclusion under the one term "reduction en masse."

¹ Obs. de Chir., Paris, 1702.

² Lond. Med. Gazette, 1828, vol. i., p. 484.

³ *Idem.*, 1850, p. 236.

⁴ Med. Chi. Trans., xlii., p. 247.

⁵ Tracts, B. 339, 1851.

Varieties that may occur in inguinal hernia.

1. *The hernial sac and its contents have been completely reduced through the internal abdominal ring into the subperitoneal tissue.* (*Vide fig. 1*). At least eighteen instances of this occur in the tables.

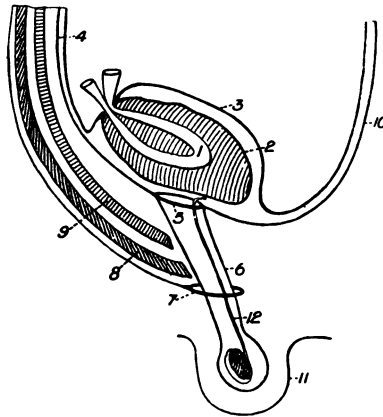


FIG. 1.—Complete Reduction en Masse.

- 1 = Small intestine.
- 2 = Sac completely reduced.
- 3 = Peritoneum pushed up.
- 4 = Transversalis fascia.
- 5 = Internal abdominal ring.
- 6 = Infundibuliform fascia.
- 7 = External ring in external oblique.
- 8 = Internal oblique.
- 9 = Transversalis muscle.
- 10 = Iliac fascia.
- 11 = Skin.
- 12 = Vas.

2. The *intestine* has been reduced from an inguinal sac into an abdominal subperitoneal sac. This variety is much the commonest. *Some of the sac still remains* in the *inguinal canal*, or even in the scrotum. The abdominal and inguinal sacs have a common neck. The subperitoneal sac may be a portion of the original inguinal sac, which has ascended with the intestine during the taxis, or it may have been gradually formed previously. (*Vide* fig. 2).

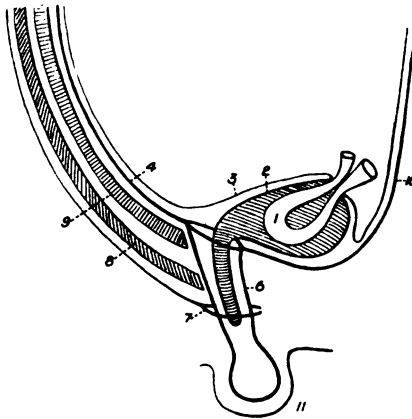


FIG. 2.—Incomplete Reduction en Masse.

- 1 = Small intestine.
- 2 = Sac.
- 3 = Peritoneum pushed up.
- 4 = Transversalis fascia.
- 6 = Infundibuliform fascia.
- 7 = External ring in external oblique.
- 8 = Internal oblique.
- 9 = Transversalis muscle.
- 10 = Iliac fascia.
- 11 = Skin.

3. The intestine has been reduced from the inguinal sac through the internal abdominal ring and then *through a laceration in the neck of the sac*. The intestine lies free in the subperitoneal tissue. This form was considered by Mr. Birkett to be very common. There are three excellent specimens of it in the Guy's Museum.* The rupture is usually on the posterior wall of the sac. (*Vide* fig. 3).

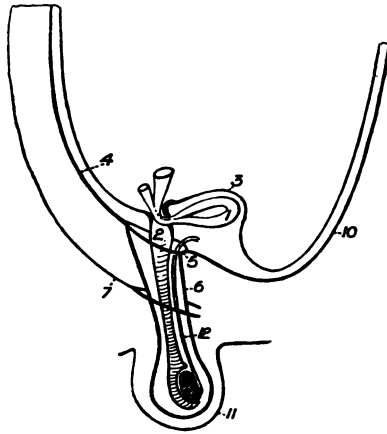


FIG. 3.—Reduction by Laceration.

- 1 = Small intestine.
- 2 = Sac
- 3 = Peritoneum pushed up.
- 4 = Transversalis fascia.
- 5 = Internal abdominal ring.
- 6 = Infundibuliform fascia.
- 7 = External ring in external oblique.
- 10 = Iliac fascia.
- 11 = Skin.
- 12 = Vas.

* Specimens 1134, 1135, 1136.

4. The intestine has been reduced into the peritoneal cavity where it lies still strangulated by a ring consisting of the detached neck of the sac. This form is very rare. (Case 13, Table I). (*Vide* fig. 4).

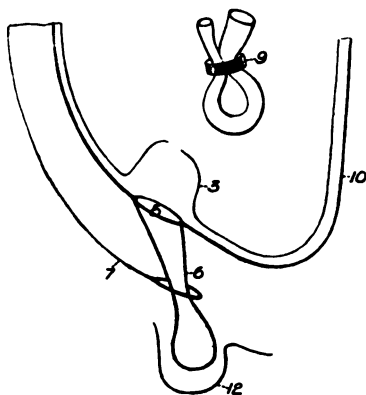


FIG. 4.—Reduction by Detachment of the Orifice of the Sac.

- 1 = Small intestine.
- 3 = Peritoneum pushed up.
- 5 = Internal abdominal ring.
- 6 = Infundibuliform fascia.
- 7 = External ring in external oblique.
- 9 = Ring orifice of sac.
- 10 = Iliac fascia.
- 12 = Vas.

5. The intestine has been reduced from a scrotal sac into the inguinal portion of an hour-glass-shaped sac. The sac has, however, ascended to some extent generally. Some would exclude this variety. It is, however, difficult to distinguish it clinically from the other forms. (*Vide* fig. 5).

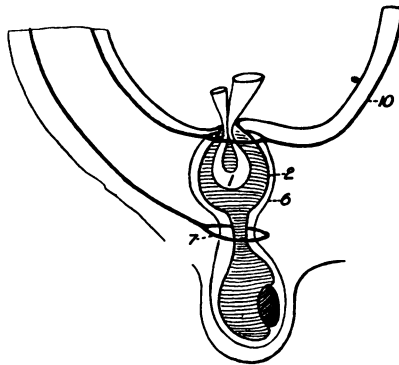


FIG. 5.—Reduction from one Compartment of a Congenital Sac into another one.

- 1 = Small intestine.
- 2 = Sac.
- 6 = Infundibuliform fascia.
- 7 = External ring in external oblique.
- 10 = Iliac fascia.

6. The testicle has been reduced with the hernia into the sub-peritoneal tissue. Case 10, Table I. (*Vide* fig. 6, a and b).

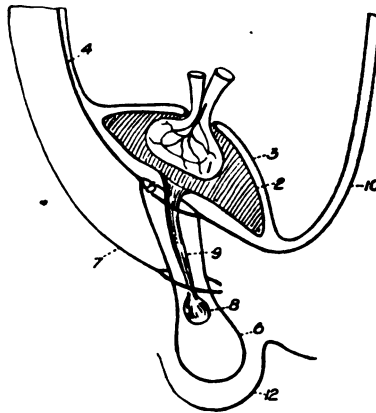


FIG. 6a.—Retraction of Testicle.

- 1 = Small intestine.
- 2 = Sac.
- 3 = Peritoneum pushed up.
- 4 = Transversalis fascia.
- 5 = Internal abdominal ring.
- 6 = Infundibuliform fascia.
- 7 = External ring in external oblique.
- 8 = Testicle.
- 9 = Cord.
- 10 = Iliac fascia.
- 12 = Skin.

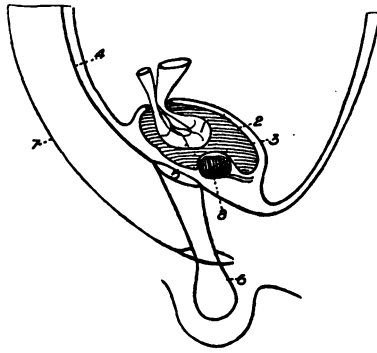


FIG. 6b.—Reduction of the Testicle into the Subperitoneal Tissue.

- 1 = Small intestine.
- 2 = Sac completely reduced.
- 3 = Peritoneum pushed up.
- 4 = Transversalis fascia.
- 5 = Internal abdominal ring.
- 6 = Infundibuliform fascia.
- 7 = External ring in external oblique.
- 8 = Testicle.

MECHANISM.

In nearly all the cases the constriction is at the neck of the sac, and all the cases in the present series are oblique inguinal herniæ. In the large majority the hernia is of long duration, the average being sixteen years. In six cases the hernia became strangulated on its first descent, and four of these (and probably all) were vaginal. It is well known that the orifice of the sac frequently gets contracted in old herniæ, and that the orifice is very small in hernia into the vaginal process, from the beginning. The wearing of a truss for a long time also causes some contraction of the neck of the sac. The accident is therefore especially likely to occur in a hernia with a *very contracted and rigid orifice to the sac*. A *very tight strangulation* from any cause is another important factor.

The rings and inguinal canal are usually dilated in these chronic cases, and they allow the ascent of a considerable elastic

tumour along them. The taxis fails to reduce the intestine through the very narrow and firm orifice of the sac, and the pressure on the tense and semifluid scrotal tumour then produces dilatation of the inguinal part of the sac. When no further dilatation of the sac can occur in the rigid canal, the pressure is almost entirely conducted up to the neck of the sac, which it separates from the transversalis fascia at the internal ring. The portion of the sac that now lies just below the orifice, placed within the subperitoneal tissues, is left with very little external support to counteract the fluid pressure within it. It therefore dilates, and the more it dilates the more it drags upon the inguino-scrotal sac, which therefore follows it. If the lower part of the sac is firmly fixed to the surrounding structures, the neck may rupture from over-distension, and allow the fluid and intestine to escape into the subperitoneal tissue. The cord and testicle may, however, accompany the sac, and even the scrotal skin became invaginated in one case of Dupuytren's. It follows that laceration of the neck of the sac is particularly likely to happen in cases of hernia into the vaginal process. Reduction of the testicle occurs in the congenital cases for the same reason, namely, the attachment of the sac to the testicle and cord. When the hernia has been reduced into the inguinal canal, it is difficult to apply powerful taxis, and it therefore follows that the cases of incomplete reduction en masse are by far the commonest. For the same reason also reduction en masse is most common in scrotal cases.

The abdominal sac may, in some cases, have been formed gradually, as suggested by Mr. Cock⁷.

The repeated forcible reduction so frequently practised by the patient may produce this gradual formation. In Case 80, Table III., the sac was completely and gradually reduced in this way. In Case 19, Table III., the sac had probably been partially reduced in a similar way. The reduction was completed after the onset of strangulation.

⁷ Guy's Hospital Reports, 1847.

The long wearing of a truss may cause the development of a diverticulum from the neck of the sac by preventing the protrusion of the intestine into the lower part of the sac, while allowing its entry into the upper part, which dilates. Case 12, Table III., may be an instance of this.

Mr. Birkett considered that a complete reduction en masse did not occur in cases of inguino-scrotal hernia. His paper served to draw the attention of subsequent operators to this point, and since then several examples have been recorded (*vide* Tables). Reduction en masse is much more common in large than in small herniæ. Although in most of the cases the force and duration of the taxis have been excessive, yet some of the accidents have been produced by gentle taxis, and there is one instance of spontaneous reduction recorded.⁸

Adhesion of the bowel to the sac may be a cause in rare cases.⁹ It is remarkable that one can find no case recorded in which the sac contained large intestine, and it is very uncommon for it to contain omentum. Mr. Birkett believed that reduction en masse was very much more common in hernia into the vaginal process; but out of the twelve inguinal cases which have occurred at Guy's Hospital since 1860, eight were acquired and only three were congenital.

It is a curious thing that a scrotal sac can be detached and reduced into the abdomen without causing any evident bruising of the tissues of the scrotum and inguinal canal; but the hernial sac has looser attachments than is generally supposed. This is sometimes well seen at the operation for radical cure.

Cloquet was able to reduce the sac on the cadaver in twenty-five cases. Some of these sacs were empty.

When the hernial sac is adherent to the skin, the latter may become invaginated to follow the sac.

In two¹⁰ of the inguinal cases the injury was inflicted *at the operation of herniotomy*, by reducing the rupture without opening the sac. This dangerous practice has been wisely abandoned.

⁸ Case 3, Table V.

⁹ Richeraud; Dict. des Sciences Med.

¹⁰ Case 11, Table I.

This accident is much more common in *herniotomy* for femoral¹¹ hernia. The sac has generally been mistaken for the bowel, and has been reduced sometimes easily, but usually with considerable difficulty. The comparative frequency of this mistake in femoral hernia is due to the peculiar difficulty of recognising the coverings of this hernia, the fascia propria having been taken for the sac. Another cause is the remarkable mobility of the sac in some of these cases. The fatty subperitoneal tissue is not uncommonly mistaken for omentum. The mistake can be avoided by reasonable care and by passing the finger up the crural canal to make sure that the intestine has reached the peritoneal cavity. It is rare for a femoral hernia to be reduced *en masse* by taxis only. The obturator case, placed at the end of Table V., was probably reduced by muscular action of the obturator externus during eversion of the thigh.

SYMPTOMS.

The general symptoms of strangulation may diminish for a time after the apparent reduction, and this may be very misleading. The constipation, however, remains absolute in almost all the cases. (In a Richter's hernia the bowels may be relieved several times. Case 8, Table IV., is an example of this.) Soon the symptoms return in an aggravated form.

The local signs. Out of the 78 cases of inguinal hernia reduced by taxis there were local signs in 55. Probably they were present also in some of the others, in which the reports make no statement on the subject. There were no local signs noticed in the *herniotomy* cases, nor in the femoral cases. The commonest local indications are the following :—

1. An ill-defined swelling or fulness in the upper part of the inguinal canal, or near the internal abdominal ring, or in the iliac fossa.
2. Pain and tenderness over the same regions.
3. Failure to reproduce the rupture by coughing and straining. This may indicate the side involved when two ruptures co-exist.
4. Repeated reproduction of the tumour after taxis.

¹¹ Tables IV. and V.

5. An unduly patent canal or external abdominal ring.
6. Retraction or disappearance of the testicle.
7. A small flaccid tumour, at the external ring, giving no impulse on coughing.
8. A tumour may be felt in the inguinal canal by the finger which has been introduced by invaginating the scrotum. This tumour may or may not give an impulse.
9. The failure to feel a scrotal sac in a case known to have been one of scrotal hernia.
10. Gurgling over the inguinal region on palpation.
11. A pelvic tumour in some femoral and in obturator cases.

DIAGNOSIS.

When a strangulated hernia has been apparently reduced but the general symptoms of strangulation persist, it is very probable that a reduction en masse has occurred. Certain other complications and sequelæ of strangulated hernia may be confused with reduction en masse, and may cause considerable anxiety and doubt in the diagnosis. Some of these are :—

1. Paralytic distension, or indentation of the properly reduced intestines.
2. Volvulus associated with an external hernia.
3. Strangulation by bands, and other forms of acute intestinal obstruction, which may coexist with a hernia.
4. Peritonitis following the reduction of a strangulated hernia.
5. Strangulation of the intestine by an omental sac, which may have been reduced from the sac of an external hernia.

In cases of enteritis there is usually severe diarrhœa to distinguish them.

Strangulation by an omental sac within the sac of a hernia is very uncommon. Complete omental sacs are uncommon except in umbilical hernia, in which reduction en masse, rarely if ever occurs.

The omental sac is generally more or less adherent to the hernial sac; and its reduction out of the latter is thus rendered unlikely.¹² No local signs of reduction en masse exist in such a

¹² Hewett; *Med. Chi. Trans.*, 1884, Vol. xxvii.

case. When peritonitis follows upon the reduction of a strangulated hernia and induces vomiting with constipation, the constipation is rarely complete as regards both flatus and fæces. The great rigidity and uniformly tender abdomen, associated with some fever usually render the diagnosis easy. In more localised peritonitis adhesions and kinking of the intestine may, however, occur, and simulate reduction en masse very closely. The absence of any local indication of reduction en masse may be of considerable importance in determining the nature of a difficult case of this kind.

When internal obstruction coexists with an external hernia, if the hernia can be easily reduced and reproduced, it is certain that some internal variety of obstruction exists. The early and great distension usually prominent in cases of volvulus may indicate the nature of that obstruction. An incompletely reducible entero-epiplocele, or an inflamed gland associated with a hernia and an internal strangulation may mislead even the wisest. Mr. Cock diagnosed reduction en masse in a case of volvulus which co-existed with a hernia and an inflamed gland.

The coexistence of an internal strangulation by a band, and a strangulated inguinal hernia is fortunately a very rare thing. Mr. Adams¹³ records a case in which, the strangulated hernia having been reduced, and the symptoms persisting, the diagnosis of reduction en masse was erroneously made. The persistence of the symptoms was really due to strangulation by a mesenteric band in the right iliac region. Case 17, Table III., is an instance of the association of a reduction en masse with an internal strangulation by a band.

Paralytic distension of the intestine reduced from a strangulated hernia may cause great difficulty in the diagnosis. They are generally cases of long duration of strangulation. There are no local indications of reduction en masse. In searching for these indications, especially in femoral cases, the pelvis should be explored from the rectum or vagina. Some flatus may be passed in cases of paralytic distension. Mr. Davies-Colley¹⁴ performed

¹³ Adams; *Med. Times and Gazette*, 1853, p. 448.

¹⁴ Davies-Colley's Reports, 1887, No. 144.

an exploratory laparotomy on a case of paralytic distension two days after a kelotomy. In considering the diagnosis it is important to remember that reduction en masse is most common in cases of right, oblique, large inguinal hernia of long duration.

Out of sixty-six cases, the hernia was on the right in forty-five cases, and on the left in twenty-one. The average duration was sixteen years. It may, however, occur in a hernia on its first descent. Nearly all the inguinal cases occur in men, and almost all the femoral cases occur in women.

Out of the hundred cases in the following Tables, eighty are inguinal, nineteen femoral, and one obturator. The patient's conviction that the hernia has not been properly reduced should not be ignored. In Case 8, Table IV., the hernia was of the Richter variety, and the bowels were opened several times after taxis had been used. The other symptoms of strangulation persisted. A pelvic examination would probably have led to a correct diagnosis in this, and also in the obturator case. In cases of double hernia, the side may be determined by the history of a difficulty in the reduction of one of the ruptures, and the failure to reproduce it by straining. Pain along the inguinal canal may also serve to indicate the side affected (Case 30, Table III.). Dupuytren, in one case, opened the right canal first, and found the sac to be empty. He then opened the left canal, and found the reduced sac, and the patient recovered (Case 2, Table II.). A most careful search should be made for local indications when the patient is under the influence of an anæsthetic and everything is ready for the operation.

PROPHYLAXIS.

It is clear that excessive taxis is the chief cause of this serious accident. The manipulation may be too forcible or too prolonged; and it is especially dangerous when the patient is under the influence of an anæsthetic.

The patient has inflicted the injury in but a few of the cases. In the following series of cases the medical man caused the "reduction" in sixty-three cases, and the patient caused sixteen only. These figures contradict the general statement that the patient has generally effected the "reduction." In the days of

septic surgery, prolonged taxis may have been justifiable. There is no excuse for it at the present day. It is rare for the patient to derive much benefit from taxis applied for more than three or four minutes. It is far better for him to undergo the operation of herniotomy than to submit to the repeated taxis and the deplorable delay which are still far too common in the treatment of strangulated hernia. Reduction en masse is only one of the many dangers of excessive taxis. Great care should be exercised to find, and to always open the proper sac at the operation of herniotomy. This is especially necessary in femoral hernia, in which operation reduction en masse is commonest. The finger should be passed up through the divided orifice of the sac to make certain that the intestine has been properly reduced.

TREATMENT.

As soon as it appears probable that a hernia has been reduced en masse an exploratory operation should be undertaken. Delay is nearly always fatal, and it is very rare in reduction en masse for an operation performed later than the fifth day of strangulation to be successful. When local indications are evident, the inguinal (or femoral) canal should be explored. This also seems to be the best course to adopt in the few cases in which no local signs can be discovered. Abdominal section has been proposed and practised by some surgeons in these cases. Annandale and Stokes have each recorded a fatal case treated by this method. The main objections to the adoption of laparotomy instead of the ordinary operation seem to be the following :—

1. The increased risk of peritonitis. It is not possible to foretell the condition of the contents of the reduced sac ; and the operator, working in the dark, may unnecessarily divide the stricture and induce peritoneal extravasation of pus, or of fæces.

2. The more severe shock which such an operation may inflict upon a patient already seriously exhausted by prolonged vomiting.

3. A radical cure of the hernia cannot be performed.

4. The difficulty of reaching the stricture and of safely dividing it from a laparotomy wound may be considerable. Mr. Stokes experienced this difficulty in his case. Mr. Annandale,

however, was more fortunate, and was able to reduce the intestine with ease without dividing the stricture.

This method has, however, some compensating advantages in some obscure cases. It allows a free exploration of the abdominal cavity, and this may be of great advantage in cases of internal strangulation which may be mistaken for reduction en masse or may complicate it. The internal strangulation can thus be found and treated without delay. Sometimes a femoral or inguinal exploration has failed to discover an existing reduction en masse, and the upper of two sacs has been mistaken for the peritoneal cavity. These mistakes could hardly have occurred if a laparotomy had been employed in these cases. The errors can be avoided also by making the inguinal incision of a sufficient size, and the search a very thorough one. A laparotomy might have saved the life of the above case of obturator hernia reduced en masse. In patients known to suffer from more than one hernia, difficulties have arisen on several occasions. Two or more herniæ may have been recently reduced after the onset of intestinal obstruction; and either no local signs may be present or there may be a misleading one. Dupuytren must have had considerable confidence in his diagnosis, when, having opened the right inguinal sac and found it empty, he explored the left inguinal canal and found his hopes realised. (Case 2, Table II.)

The inguinal exploration.—The external ring should be exposed by the usual incision. A sac may in some cases be discovered at the external ring, and it may be possible, to pull down the reduced portion of the sac, to open it, to divide the stricture and reduce the intestine. A finger introduced into the inguinal canal may feel the sac, which may be seized with forceps and drawn down. It is generally necessary and always wise to open the inguinal canal freely in order thoroughly to expose the sac. It may be necessary to prolong the incision upwards and outwards through the lower fibres of the internal oblique and transversalis muscles before the sac can be discovered. The incision should be free enough to allow of a proper exploration of the parts. The operation is a difficult one even through a free incision, and it is disastrous

to attempt it through a small one. This false conservatism was the chief cause of death in eleven out of the thirty-one fatal cases following operation. (Table III.). The sac should be opened with more than the usual precautions, because adhesion of the intestine to it is not uncommon. The contained fluid should be swabbed away because it is very liable to be infective. The real orifice of the sac can be discovered by passing the finger upwards, along the anterior surface of the mesentery. The stricture should be divided with care and the bowel loop drawn down to examine its neck. If a greyish patch be discovered, it should be protected either by oversewing or by grafting a piece of the sac over it, after the manner successfully adopted by Mr. Dunn. (Cases 35, 36, Table II.)

The operation may be completed by performing a simple form of radical cure if the condition of the patient permits the necessary extension of the time.

When the intestine has been ruptured by taxis, or has perforated into the sac, it may either be left in the sac, or resected. Of the two the former is probably the safer method of treatment for these patients, because they may be too ill to survive an immediate resection. It may be necessary to carefully incise the stricture to allow a free evacuation of the contents of the distended bowel above, but the sac should be thoroughly cleansed first. This seems to be of especial value when the perforation is a small one located at the fundus of the loop. The fæces may then pass along the intestine instead of through the perforation into the wound. This, I think, accounts for the spontaneous closure of the fæcal fistula in my case. A risk of peritonitis attends this division of the stricture. The stricture is, however, much tighter than that observed in the ordinary cases of strangulated hernia. If the perforation is a large one and the finger can be passed through it into the lumen of the intestine above the structure, the latter may be left undivided. There is little to gain by incising the stricture in such a case, and the risk of peritonitis prohibits it. At a later date an operation will probably be necessary to close the fæcal fistula.

It is extremely uncommon for recovery to occur without operation. Sanson¹⁵ has described a case which may possibly have been an instance of this. His case was treated by repeated enemata; after several days the hernia re-descended. Case 2, Table V. is another doubtful case in which a fæcal fistula followed a supposed reduction en masse.

In Case 22, Table II., the surgeon induced the patient to cause a re-descent of a probable reduction en masse by straining and coughing. The surgeon then secured a part of the sac with his left hand, and reduced the bowel with the right. I am afraid that such treatment is not likely to be often attended with success; and it is not even by any means certain that this case was really one of reduction en masse.

PROGNOSIS.

The prognosis is very serious. Out of the one hundred cases tabulated below there were fifty-nine deaths and forty-one recoveries. Of the eighty inguinal cases, thirty-six recovered and forty-four died. Thirty-five recovered after an operation, and one without an operation. Thirteen cases died untreated by operation, and thirty-one after an operation.

In nineteen femoral cases there were fourteen fatalities and five recoveries. Six died, although an operation was performed, and eight without operation. One recovered without operation, and four after an operation. The only obturator case was fatal. It will be noticed that the prognosis of femoral cases is more grave than that of the inguinal. This is probably due to the absence of local signs, or failure to recognise them, in femoral cases. Delay in treatment is therefore more common, and the operations are less thorough. It is almost certain that the mortality is higher than the figures indicate, because only the successful cases are often recorded. This seems to be clear from the fact that, of fifteen previously unreported cases included in the Tables, there were only five recoveries. One of the fatalities was due to an accidental cause, however; and three of the others were femoral cases. With a recognition of the facts that the treatment must be prompt, and that the operation must be

¹⁵ Diet de Med. et de Chir, 57.

thorough to be successful, better results may be hoped for in the future.

The commonest causes of death are peritonitis, exhaustion from long continued strangulation, and septic infection of the wound.

FULL REPORT OF CASE 84.

E. L., a man of 55 years, came into Bright ward on April 6th, 1898, suffering from thrombosis of the left internal saphenous vein. He also had an acquired right scrotal hernia, which had first appeared six months before admission. An inefficient truss had been used. The patient had experienced great difficulty in reducing the hernia on several occasions. During the night of April 10th, the hernia again descended and became strangulated. The patient made several vain attempts to reduce it. Next morning I reduced the hernia fairly easily. The patient went home a few days later.

On May 8th, 1898, the hernia reappeared and became strangulated. The patient repeatedly tried and failed to reduce it. Next day a doctor failed to reduce it. On the 10th, after prolonged and very painful taxis, the doctor reduced the rupture. The patient, however, felt convinced that the reduction was not satisfactory. The symptoms, although relieved for a time, became worse. Vomiting became severe and frequent, constipation remained absolute, and the abdomen became distended. Colicky pains worried the patient at short intervals.

The man was admitted into Bright on May 11th, at 11 p.m. He then looked very ill and collapsed, with a pulse of 120 and feeble. His face was pinched and leaden and the eyes sunken. The abdomen was uniformly distended and tender. There was an abnormal fulness over the right inguinal canal. A vague tender swelling was felt above the internal abdominal ring, which was much more distinct later under an anæsthetic. The external ring was large and well defined. The testicle was not retracted.

A.C.E. was at once administered. The inguinal canal was opened freely and a sac was found high up in it. The sac was

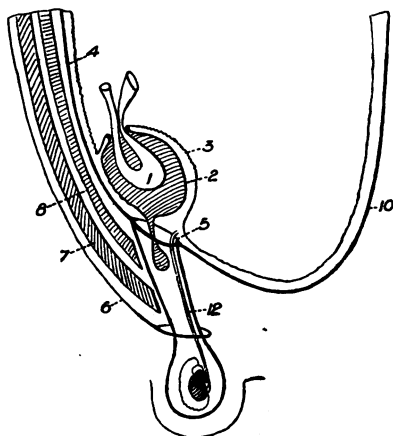


FIG. 7.—Illustrating Case of E. L.

- 1 = Small intestine.
- 2 = Sac in subperitoneal tissue.
- 3 = Parietal peritoneum pushed up.
- 4 = Transversalis fascia.
- 5 = Internal abdominal ring.
- 6 = External oblique with external ring.
- 7 = Internal oblique.
- 8 = Transversalis.
- 9 = Infundibuliform fascia.
- 10 = Iliac fascia.
- 11 = Small sac in inguinal canal.
- 12 = Vas.

incised, allowing some gas and fæcal fluid to escape. The sac wall was very thick. On passing my finger upward I felt a tubular constriction of the sac just within the internal abdominal ring. The finger was passed through this constriction with difficulty into another and much larger sac lying entirely above the internal ring and containing strangulated small intestine. No attempts availed to bring this sac down into the wound. The incision was therefore extended upwards and outwards through the abdominal muscles for two inches above the internal ring. The second sac was thus partly exposed, and was incised. About six inches of small intestine, much congested and covered with recent lymph. were thus partly exposed. The intestine was adherent to the sac in several places. The sac was well irrigated and dried with iodoform gauze. The neck of the sac was then discovered near the anterior abdominal wall three inches above the internal

ring. The constriction, which was a very tight one, was carefully incised. No attempt to find the perforation was made, lest extravasation of fæces into the peritoneal cavity should be induced. The loop of intestine was left in the upper sac, and the lower sac was removed. The lower part of the wound was sewn up with catgut, and a gauze drain was left leading from the loop of intestine into the upper end of the incision. Some fæces escaped for about three days and the wound suppurated, but it gradually healed. The patient was discharged six weeks after the operation wearing a specially designed blade-truss. This he has worn since.

He has remained quite well for nearly three years. At the present time there is no evidence of the existence of a hernia of any kind in the right inguinal region. The patient, however, tells me that he has suffered from several mild attacks of intestinal obstruction during the summer. He also says that in August he had complete constipation for eight days. During this attack there was some swelling, pain, and tenderness in the right inguinal region. The symptoms were relieved by the persistent use of enemata.

This case seems to be worthy of publication for several reasons :—

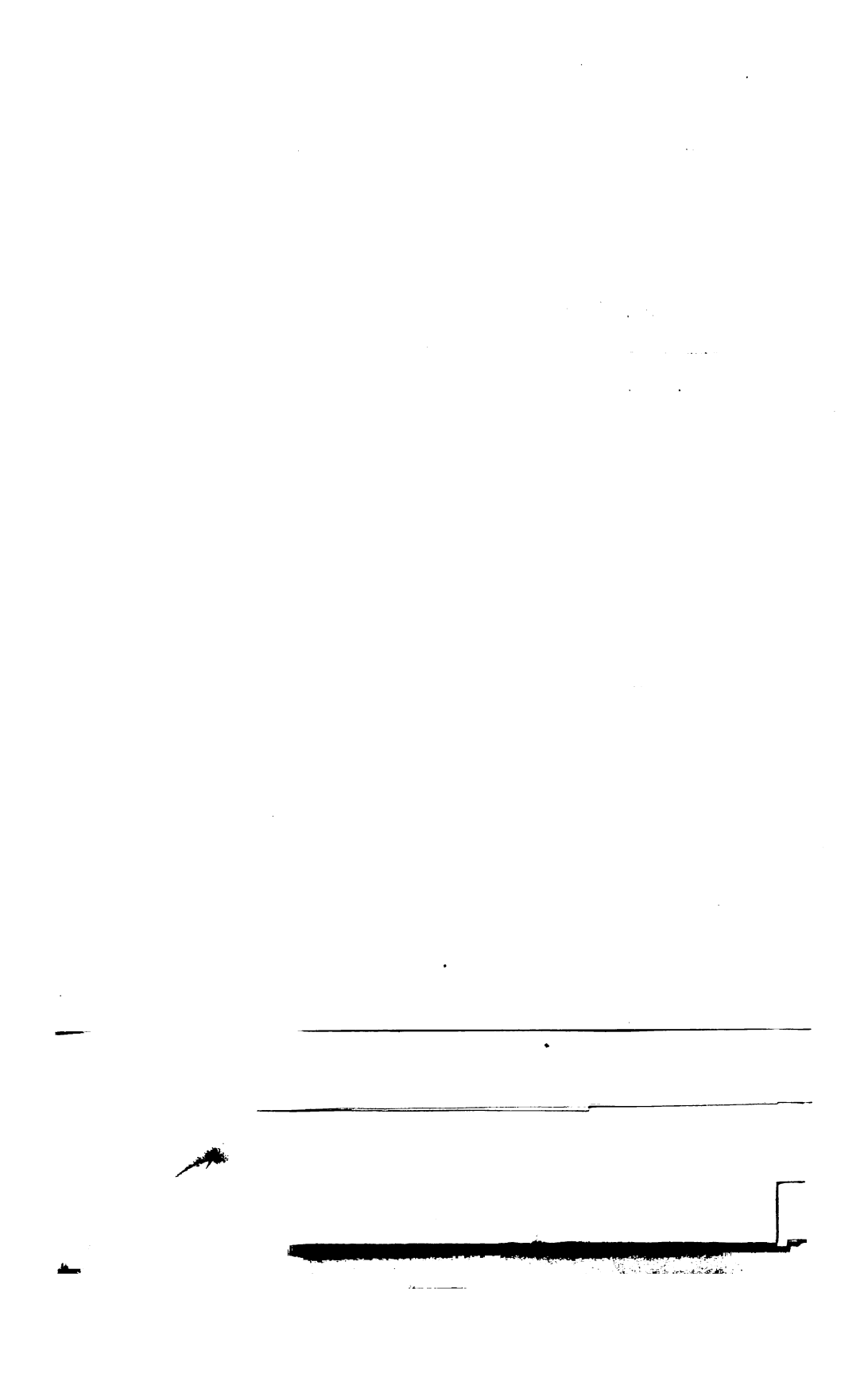
1. *The intestine had probably been ruptured within the sac during the taxis.*

2. *It is the only case I can find recorded where the stricture has been divided and the ruptured intestine has been successfully left in the retroperitoneal sac.* This was considered to be better than resection in this case. Resection and reduction of the intestine would probably have caused peritonitis, for the sac contained fæces, and was already suppurating. Moreover, the patient was too ill to successfully undergo a resection at the end of a tedious operation. That the fæcal fistula should have spontaneously healed was, of course, unexpected.

3. *It would have been disastrous to have treated this case through a laparotomy wound, and it indicates some of the evident advantages of the inguinal over the abdominal operation in these cases.*

nal Hernia, r

Survived this attack.		Anno.	Reference.
Reduction 6 months. n, contained Last severe attack, 8 days.		1740	Arnaud Diss. p. 387, Obs. ix.
—	ing, where it	1818 1819	Scarpa Sull'ernie, 2nd edit., p. 49.
about 3 days	... rom internal	1828	Sir Charles Bell.; Lon. Med. Gazette, 1828, vol. i., p. 484.
—	Idem	Idem.
—	1834	B. B. Cooper; Guy's Hosp. Rep. vol. IV., p. 331.
to 7 days	1836	Parish; Observ. on Strang. Hernia.
—	1840	Langier; Bull. Chir., i., 363.
about 4 days	1842	Banner; Prov. Med. and Surg. Journal, 1843.
about 4 days	...	1844-5	Blackman, p. 38.
Died third day of strangulation rather suddenly.	1877	Howse; Reports.
days	... ssue ...	1880	Clement Lucas; Reports.
days	... tumour was n the canal.	—	McCarthy; Trans. Path. Soc., 1881, p. 80.
days	... the sac had drium.	—	Idem.



ON ENTERIC FEVER:

BEING

AN INVESTIGATION INTO THE BACTERIOLOGICAL
CONDITION OF THE URINE, AND IN SOME CASES,
OF THE KIDNEY IN THIS DISEASE.

(THESIS FOR THE M.D. CAMBRIDGE.)

By STANLEY E. DENYER, C.M.G., M.A., M.D., B.C., D.P.H.,

From the Bacteriological Laboratory, Guy's Hospital.

IN bringing before you this paper on the result of some investigations into the bacteriology of the genito-urinary tract in enteric fever, I will not venture to apologise for its shortcomings, nor for its inadequacy, because of these I am fully conscious. The subject is one which demands more attention than has hitherto been bestowed upon it, both from a clinical, pathological, and public health point of view. Very little help is to be obtained from the literature, which at present is but scanty. The earlier publications are of little use, since our bacteriological knowledge was not then sufficiently advanced to satisfactorily differentiate the micro-organism found. Krogius, writing on the subject of bacteriuria, bases his remarks upon eight cases, in which he examined the urine under aseptic conditions. In each of these cases he states that he found the bacillus coli communis. He has, however, given but scanty proof of the identity of the organism found. For example, in

Observation II. his proof of the presence of the bacillus coli communis is given in the following words: "l'ensemencement de l'urine sur des plaques de gélatine, donna lieu au développement d'innombrables colonies du bacterium coli commune," and in Observation III. "Sur les plaques de gelatine, il se développee le bacterium coli commune en culture puré." In none of the cases can I find that he published any further proof of the identity of the organism found, although he is quoted as having made most careful bacteriological examinations.¹ In another case, published as late as 1898, in speaking of the bacillus coli communis, the author says, "the bacteria were motile and otherwise characteristic."² The later publications seem to shew that the urine of patients suffering from enteric fever contained the bacillus typhi abdominalis in about twenty-five per cent. of cases. In the few cases that I have examined, the results have shown a lower percentage than this.

My own object has been to examine into the bacteriological condition of the urine in enteric fever, from the earliest possible period in the disease, to carry on this investigation during the whole period that the patient remained in hospital under treatment, and in cases where death has occurred, to make examinations of the viscera as early as possible after death. Bacteriological examinations were made of the heart-blood or spleen, the kidneys, and the urine in the bladder. Sections of the kidneys were also made and examined for micro-organisms, with the idea of finding, if present, their position in these organs, and whether in the tubules, glomeruli, or in both.

From the small number of cases that I have been able to examine, I am afraid that any deduction would be presumptuous. But I think, as far as it is possible, under the circumstances under which these examinations can be made, that the few facts I have gleaned are reliable.

¹ A. Krogius. Sur la bacteriurie, Annales des Organ. Gén-Urin., 1894, p. 196, et seq.

² Nathan, P. W. Bacillus coli communis in the urine, and its significance. Med. Rec., New York, 1898, vol. liii., 83-86.

METHODS ADOPTED IN THE BACTERIOLOGICAL
EXAMINATION OF THE URINE, KIDNEYS AND OTHER
VISCERA, AND THE BLOOD, IN ENTERIC FEVER.

The Bacteriological Examination of the Urine.—In the case of male patients the glans penis and meatus urinarius were washed with a solution of mercuric chloride (1—2000), or with lysol 2 per cent. The urine was then passed (the first portion being rejected, as likely to contain organisms that might be present in the urethra) into a sterilized flask, which was immediately closed with its sterilized plug of cotton wool.

In the case of female patients, the urine was drawn off by a catheter, passed by a reliable nurse, the parts being first cleansed with a solution of mercuric chloride (1—2000). Some of these catheter specimens obtained by the nurse in this way were found to be quite sterile. I have mentioned this fact, in order to point out that the specimens obtained in this manner can be relied upon equally with those obtained from male patients.

In each case hanging drop examinations of the urine were made. 5 c.c. of the urine were pipetted, with a sterile pipette, into tubes of broth and glucose-formate broth respectively. An agar plate was made and some urine spread over the surface, every means being adopted to prevent the entrance of adventitious organisms. The glucose-formate broth tube was incubated in every case anærobically, a Buchner's tube being used.

All three media were incubated for twenty-four hours and two days at 37° C. At the end of these times they were examined, hanging drop preparations being made, and films being stained with carbol-methylene blue, and if bacilli were found, by Gram-Weigert's method.

Agar plates were then made, and if more than one organism was present, separation was thus effected. In some cases it was found necessary to replate several times, in order to obtain the organisms in pure culture.

When micrococci only were found, they were not worked out. When pure cultivations of bacilli were found, the organism was worked out according to the following scheme:—

Description.—Whether coccus, bacillus, etc.

Motility.—Whether non-motile, slightly motile, very motile, etc.

Spores.—Presence or absence of.

Staining reactions.—With carbol methylene blue, carbol fuchsin, Gram-Weigert.

Pleomorphism.—Any signs noted.

Cultural reactions.—The growth of the organism in broth and peptone water. These were examined at the end of five days for the production of indol, recognised by the fact that when acted upon by nitric acid in the presence of nitrites, a nitroso-indol compound is produced, which has a very red colour. (By using commercial nitric acid, which contains nitrous acid as an impurity, this experiment can easily be performed.)

The reactions of the organism when grown upon lactose broth,^a formate broth, dextrose broth, saccharose broth, glycerine broth, nitrate broth, lead broth (for detection of sulphuretted hydrogen), litmus milk, and glucose-formate broth grown anaerobically.

The characters of the growth upon agar, blood-serum and potato. All of the above were incubated at a temperature of 37° C. The growth of the organism upon gelatine (stab, streak, and plates), at a temperature of 20° C., was also investigated, and the presence or absence of gas-production, or of liquefaction was especially noted.

All were examined at the end of twenty-four hours, and at the end of two, three and five days. An examination was made earlier than twenty-four hours, in dextrose broth cultures for gas-formation, and motility, and later than twenty-four hours in the case of milk for the occurrence of clotting.

Bacteriological examination of the kidney.—The following procedure was adopted in the bacteriological examination of the

^a Lactose broth, dextrose broth, formate broth, saccharose broth and glycerine broth, as well as litmus milk, were coloured blue with litmus, any formation of acid being indicated by a change in colour.

kidney. The kidney was removed from the body as soon as possible after death, the capsule was then stripped off, and the surface of the kidney seared with a hot iron and thus sterilized; an incision was next made with a sterilized knife into the kidney substance, and a scraping was taken with a sterilized platinum wire loop, and planted in broth and glucose-formate broth, and on sloped agar. Direct films were also made, stained, and examined for micro-organisms. The media after inoculation were incubated at 37° C. for twenty-four hours and then examined. Hanging drop preparations were made and examined. Films were stained with carbol-methylene blue, carbol-fuchsin, and by Gram-Weigert's method.

Plate preparations on agar were now made, and if more than one organism was found, separation was thus effected. The same routine method of working out the organisms was adopted in all the cases.

The bacteriological examination of the spleen was carried out in a similar manner.

In the bacteriological examination of the heart-blood, the pericardium was opened, and the surface of the heart sterilized with a hot iron. A sterilized pipette was plunged through the wall of the ventricle, and some blood sucked into it. The fine end was then hermetically sealed, until the blood thus obtained was required for cultivation.

In the bacteriological examination of the urine from the post-mortem bladder, the surface of the bladder was sterilized with a hot iron, an incision was then made into it, sufficiently large for the introduction of a sterilized pipette, some urine being withdrawn, collected in a sterilized flask, and examined as described above.

Histological examination of the kidney.—In each case the kidney was examined as soon as possible after death, both to avoid, as far as one could, the presence of micro-organisms due to post-mortem changes, and the post-mortem changes in the kidney cells.

A suitable portion of the kidney was hardened, by placing it for twelve hours in a saturated solution of mercuric chloride. It

was then immersed in equal parts of spirit and water for a few hours, then in spirit for a few more; after that it was dehydrated with absolute alcohol, and cleared with xylol. From this it was transferred to a paraffin bath, heated to 56° C. for twenty-four hours. The portion of the kidney was then embedded in paraffin, and sections were cut with a microtome. These were stained with carbol-thionin blue, and examined carefully for micro-organisms under a one-sixth inch lens and under a one-twelfth inch oil immersion lens.

BACTERIOLOGICAL REACTIONS OF THE BACILLUS TYPHI ABDOMINALIS.

The following reactions have been taken to indicate the presence of the bacillus typhi abdominalis.

A bacillus, which is motile, which stains with carbol-methylene blue, and which is decolorized by Gram-Weigert's method. Which gives no indol reaction when cultivated on broth and peptone water, and tested with nitric acid in the presence of nitrites.

Which gives rise to no gas-formation when grown in formate broth, dextrose broth, lactose broth, saccharose broth, glycerine broth, or glucose-formate broth; this last grown anærobically.

Which produces nitrites when grown in nitrate broth, as shewn by producing a ruby-red colour when tested with metaphenylene diamine.

Which produces neither gas nor liquefaction when cultivated on gelatine; which, when grown on sloped gelatine, produces a whitish semi-translucent growth, with dentate edges.

Which gives a whitish semi-translucent growth on agar. Which produces slight acidity, but no clotting of milk. Which produces no spores. The non-production of spores is proved by exposing a twenty-hours' broth culture to temperature 80° C. for twenty minutes, and inoculating from this with a negative result. Of which a twenty-four hours' growth on broth becomes clumped when examined with serum from a typhoid patient, which has

caused clumping of a known bacillus typhi abdominalis grown in the same way. (Gruber's reaction).

By an atypical typhoid bacillus is meant an organism which differs from a typical typhoid bacillus in one principal reaction, while agreeing in other respects.

By a para-typhoid bacillus, an organism which differs from a typical typhoid bacillus, in two principal reactions, while agreeing in other respects.

The bacilli described in this paper as atypical bacillus coli communis and para-colon bacillus differ from the normal bacillus coli communis according to the same rules.

BRIEF ABSTRACT OF CASES.

Fifteen cases of enteric fever were examined. In one of these the kidney only was examined. In ten cases the urine only was examined, several examinations being made in each case. In three cases in which death occurred, the kidney, spleen, or heart-blood, and the urine from the post-mortem bladder were examined. In one case the urine was examined during the period of convalescence only.

CASE 1.—F., 28 years. Signs: Fever, enlargement of spleen, rose-red spots on abdomen, bronchitis, feeble pulse. Death seven days after admission. No Widal reaction performed. Post-mortem signs of enteric fever found. Kidney sterile. Histological examination, nil.

CASE 2.—M., 31 years. Bacteriuria occurred for ten months after an attack of enteric fever; it was arrested by urotropin. Four examinations of the urine were made during a period of three months. The bacillus coli communis and an atypical bacillus coli communis were found. After administration of urotropin, the urine became sterile.

CASE 3.—F., 9 years.—Enteric fever with relapse. Signs: Fever, enlargement of spleen, spots on abdomen, and a positive Widal reaction. Urine examined on thirty-fifth day (eighth day of relapse), sterile. Examined on thirty-ninth day, staphylococci.

CASE 4.—M., 21 years. The history points to the fever on admission being due to relapse, after a primary attack of a fortnight's duration. Signs: Fever, diarrhoea, and fulness of abdomen. Widal's reaction present. Urine examined three times during first, second, and fourth weeks. Staphylococci only on each occasion.

CASE 5.—F., 32 years. Signs: Fever, rose-coloured spots on abdomen, diarrhoea, a palpable spleen, some signs of bronchitis and a marked Widal reaction. Urine examined on five occasions, on the twenty-fifth day (sterile), twenty-seventh day (staphylococci), thirty-fourth day (staphylococci), forty-first day (sterile), fifty-fifth day (cocci).

CASE 6.—M., 27 years. Signs: Fever, diarrhoea, pain in head and abdomen, rose-coloured spots, dicrotic pulse, signs of bronchitis, and a positive Widal reaction; urine examined twice (during convalescence), staphylococci only found.

CASE 7.—M., 29 years. Signs: Fever, spots, and palpable spleen. Death occurred from hæmorrhage of the bowel during a probable relapse. Widal's reaction negative. The post-mortem signs of enteric fever were found. Bacteriological examinations were made of the spleen, kidney, and urine from the post-mortem bladder. The bacillus typhi abdominalis was found in pure culture in spleen. The kidney was sterile. The urine from the post-mortem bladder contained only staphylococci.

CASE 8.—F., 4 years. Signs: Fever, diarrhoea, fulness of abdomen, rose-coloured spots, and a positive Widal reaction. Five examinations of the urine were made (1) on the eighth day, a para-colon bacillus, (2) on the fifteenth day, a bacillus resembling in many respects the bacillus enteritidis of Gärtner, (3) on the twentieth day the bacillus typhi abdominalis, (4) on the thirtieth day, the bacillus proteus vulgaris, (5) on the forty-seventh day, both the bacillus coli communis and bacillus proteus vulgaris.

CASE 9.—M., 16. Signs: Fever, typhoid spots, constipation, spleen not palpable and a positive Widal reaction. Six examinations of the urine were made, on the thirteenth, eighteenth, twenty-first, twenty-fourth, twenty-seventh, and fortieth days. On five occasions staphylococci were found, and on the sixth, cocci which were arranged in pairs.

CASE 10.—M., 11. The temperature after being raised for three weeks became normal, but a relapse occurred with pyrexia of slight degree continuing for another three weeks. Spleen palpable, spots present on abdomen. Widal's reaction present. Urine examined four times, on the twenty-fifth, twenty-ninth, thirty-ninth, and forty-seventh days, respectively. Staphylococci only were found.

CASE 11.—M., 25 years. Signs: Fever, diarrhoea, enlargement of spleen, absence of spots, fulness of abdomen. Widal's reaction was present. Three examinations of the urine were made. On the first examination a para-typhoid bacillus found, on the second, staphylococci, on the third, a bacillus of the proteus group.

CASE 12.—F., 30 years.—Signs: General malaise, fever, diarrhoea and constipation alternating, enlargement of spleen, rose-coloured spots on abdomen. The blood was negative to Widal's reaction. Urine examined on four occasions: On twentieth day bacillus typhi abdominalis with staphylococci; on forty-third day bacillus typhi abdominalis in pure culture; on forty-seventh day cocci and some large bacillus, not the bacillus typhi abdominalis; on fifty-first day bacillus typhi abdominalis, with a proteus-like organism.

CASE 13.—M., 19 years; a severe case, with fever, headache, delirium, fulness of abdomen, rose-coloured spots. The spleen was not palpable. Widal's reaction negative. Death occurred from perforation. Bacteriological examination of the (post-mortem) urine, spleen and kidney, showed the presence in each, in pure, culture of a para-colon bacillus.

CASE 14.—M., 34 years. Signs: Fever, alternating diarrhoea and constipation, palpable spleen, rose-coloured spots on abdomen. Death occurred from hæmorrhage. Urine from bladder (post-mortem) was sterile. No satisfactory bacteriological examination could be made of the spleen and kidney.

CASE 15.—M., 11 years. Signs: General malaise, fever, slight fulness of abdomen, palpable spleen, and suspicious spots on abdomen. Widal's reaction present. Urine examined on three occasions. Staphylococci only found. The blood was examined bacteriologically and found to be sterile.

DISCUSSION OF CASES.

Forty-five complete bacteriological examinations of the urine were made. Of these, forty-two were made from the urine during life, and three were from the bladder after death.

The earliest examination of the urine was made on the eighth day of the disease, the bacillus para-coli communis being found. The earliest day on which the bacillus typhi abdominalis was found in the urine, was on the twentieth day of the disease (in two different cases of enteric fever, it was found on the twentieth day). The latest recorded examination was made on the fifty-first day of the disease, the bacillus typhi abdominalis being found, together with an organism giving the reactions of the proteus vulgaris. The latest day on which the bacillus typhi abdominalis was found in the urine, was therefore the fifty-first day of the disease. Since patients leave the hospital during convalescence, no attempt has been made to discover the period during which the bacillus typhi abdominalis may remain in the urine. A glance at Case 2 will show, however, that bacteriuria (in this case due to the bacillus coli communis) may continue for several months after the temperature has become normal.

The organisms found in the urine, in enteric fever patients, include the following:—

	Organism found.	No. of examinations of urine in which the organism was found.
1	Bacillus Typhi Abdominalis	4
2	" Para-typhi Abdominalis	1
3	" Coli Communis	3
4	" " (atypical)	4
5	" Para-coli Communis	2
6	A bacillus resembling in many respects the Bacillus Enteritidis of Gärtner. ...	1
7	Bacillus Proteus Vulgaris	3
8	Staphylococcus	23
	Total	41
	No. of examinations in which urine was found to be sterile.	5

NOTE 1.—In some cases two different organisms were found in the same specimen of urine.

NOTE 2.—The condition of the urine showed in most cases but little departure from the normal. At most but a slight trace of albumin was present, and in all the urine was acid.

In three cases, a bacteriological examination of the kidney was made on post-mortem examination.

In the first case (Case 1) the kidney only was examined, and was found to be sterile.

In the second case (Case 7), the kidney was found to be sterile, and the spleen contained the typhoid bacillus in pure culture. The urine in this case was unfortunately not examined during life. A specimen taken from the post-mortem bladder contained staphylococci only.

In the third case (Case 13), the kidney and spleen, as well as a specimen of the urine from the post-mortem bladder, were examined. All contained the para-colon bacillus in pure culture. The urine in this case had not been examined during life.

In fourteen cases bacteriological examinations were made of the urine.

A.—*In ten cases bacteriological examinations were made during the course of the disease, or during the period of convalescence immediately following :—*

In the first case of this series (Case 3), two examinations were made, the first on the thirty-fifth day (eighth day of relapse), when the urine was found to be sterile; the second, on the thirty-ninth day, when the urine was found to contain staphylococci.

In the second case (Case 4), three examinations were made during the first, second and fourth weeks. Staphylococci only were found.

In the third case (Case 5) five examinations were made, on the twenty-fifth day, when the urine was found to be sterile, on the twenty-seventh and thirty-fourth days, when staphylococci were found, on the forty-first day, when the urine was found to be sterile, on the fifty-fifth day, when cocci were found.

In the fourth case (Case 6), two examinations were made during convalescence, in both of which staphylococci were found.

In the fifth case (Case 8), five examinations were made. The first on the eighth day, on which the bacillus para-coli communis was found. The second, on the fifteenth day, in which a bacillus was found, resembling in many respects the bacillus enteritidis of Gärtner. The third, on the twentieth day, on which the bacillus typhi abdominalis was found in pure culture. The fourth, on the thirtieth day, when the bacillus proteus vulgaris was found. The fifth on the forty-seventh day, when both the bacillus coli communis, and the bacillus proteus vulgaris were found.

In the sixth case (Case 9), six examinations were made, on the thirteenth, eighteenth, twenty-first, twenty-fourth, twenty-seventh and fortieth days of the disease respectively. In the first five examinations staphylococci were found; in the sixth, cocci, chiefly in pairs.

In the seventh case (Case 10), four examinations were made, on the twenty-fifth, twenty-ninth, thirty-ninth and forty-seventh days of the disease respectively. On each occasion staphylococci only were found.

In the eighth case (Case 11), three examinations were made. At the first examination, made during the interval between the primary attack and a relapse, the bacillus para-typhi abdominalis, and also staphylococci, were found. At the second examination, made on the day of the rise of temperature due to relapse, staphylococci alone were found. At the third examination, made on the eighth day of relapse, staphylococci and an organism of the proteus group were found.

In the ninth case (Case 12), four examinations were made, on the twentieth day, when the bacillus typhi abdominalis and staphylococci were found, on the forty-third day, when the bacillus typhi abdominalis was found in pure culture, on the forty-seventh day, when cocci and some large bacilli (not the bacillus typhi abdominalis) were found, and on the fifty-first day, when the bacillus typhi abdominalis and a proteus-like organism were found.

In the tenth case of the series (Case 15), three examinations were made during the third and fourth week of the disease. Staphylococci only were found on each occasion.

B. *In one case (Case 2) bacteriological examinations of the urine were made only some months after the occurrence of the disease.*—Five examinations were made, the first seven months after the attack, the bacillus coli communis being found in pure culture. The second a month later, when an atypical bacillus coli communis was found, also in pure culture. The third, a month after this, when the normal bacillus coli communis was again found in pure culture. The fourth, a month later (ten months after attack), when the atypical bacillus coli communis was again found. After this six ten-grain doses of urotropin were administered, three times a day, for two days. The urine was examined twenty-four hours after administration of the last dose of urotropin, and was found to be sterile. There was no albumin in the urine at any time.

C. *In three cases bacteriological examinations were made from the urine, obtained from the post-mortem bladder. Unfortunately in neither of these cases had examinations been made during life.*

In the first case (Case 7) staphylococci only were found. (The kidney was sterile, the spleen showed the presence of the bacillus typhi abdominalis in pure culture.)

In the second case (Case 13) the bacillus para-coli communis was found in pure culture. (The kidney and spleen also showed the presence of the bacillus para-coli communis in pure culture.)

In the third case (Case 14) staphylococci only were found. (The kidney was sterile and the spleen showed the presence of the bacillus typhi abdominalis in pure culture.)

Proportion of cases of enteric fever in which the bacillus typhi abdominalis was found in the urine. Also the proportion of cases of enteric fever in which some other organisms were found.

In fourteen cases of enteric fever, bacteriological examinations of the urine were made, either during the fever, or during the period following the fall of the temperature, in one case extending to a period of ten months, or from a specimen of urine obtained from the bladder at the post-mortem examination (three cases).

In two cases (14·28 per cent.) the bacillus typhi abdominalis was found. In eight cases (57·14 per cent.) staphylococci (alone) were found. In eleven cases (78·57 per cent.) staphylococci alone, or with some other organism, were found.

The large proportion of cases in which staphylococci were found, lends colour to the suggestion of its being due to accidental contamination, notwithstanding every precaution possible being taken to obtain uncontaminated specimens. This is noticeable in Case 10, a male patient, aged 11 years, where foreskin was long and could not be drawn back, and which made it impossible to cleanse the meatus properly. It was not thought advisable to use a catheter. On reference to the table it will be seen that staphylococci were present on every occasion.

In Case 15, a male patient, aged 16 years, the same condition was present, and in this case also staphylococci only were present at each examination.

The *bacillus typhi abdominalis*, therefore, was found in only 14 per cent. of cases, and in these only at intervals, other organisms, closely approaching it in their bacteriological reactions, being also found. Thus, on reference to Case 8, it is seen, that an atypical *bacillus coli communis* was found on the eighth day of the disease, that a week later another bacillus, somewhat resembling the *bacillus enteritidis* of Gärtner, and that a week after this the *bacillus typhi abdominalis* in pure culture was found.

Again, in Case 11, the *bacillus para-typhi abdominalis* was found on one occasion, while on another a bacillus of the proteus group was found. It would appear from this, that although the *bacillus typhi abdominalis* is found in the urine of enteric fever patients, that other organisms, approaching it more or less closely in bacteriological reactions, are also found, and in the urine of the same patient.

Arguing from these premises, I would suggest, that if the organisms found in the urine of enteric fever patients be carefully and fully worked out, the percentage in which the *bacillus typhi abdominalis* is found, will be lower than that usually given.

In thirteen of the cases Widal's reaction was performed in the ordinary course of hospital routine. The following method was adopted. It is described by Mr. W. C. C. Pakes, Bacteriologist to Guy's Hospital, to whom I am indebted for the notes of the Widal reactions, and is to be found in an article entitled "Widal's Reaction," in Guy's Hospital Reports, Vol. LV. :—

"The serum is collected in special pipettes, made so that when the blood is collected, it can be allowed to coagulate in the middle, so that the serum can separate, and run down into the finer part, free from corpuscles."

"*The dilution of the serum.*—In order to dilute the serum, small test-tubes, and small graduated pipettes are used. Ten cubic millimetres of serum are pipetted into a small test-tube, and are mixed with 90 cubic millimetres of broth, which is measured in a second pipette. This gives a fluid containing 10 per cent. of serum: 10 cubic millimetres of this 10 per cent. serum, are all pipetted into a second test-tube, and are mixed with 90 cubic millimetres of broth. This gives a fluid containing 1 per cent. of serum. Sterile broth is used instead of water to prevent the laking of the red discs. It was shewn soon after the reaction was discovered that the red discs contained more of the agglutininus than the clear serum, and if sometimes laked blood is present, and at other times no blood, the observations cannot be said to be made under identical conditions, and the value of the reaction as a diagnostic test will be impaired."

"*The culture of the bacillus typhi abdominalis.*—The bacilli are inoculated on agar every fortnight, and grown at 20° C. As cultures are required for the reaction, broth tubes are inoculated from the most recent agar culture, about eighteen hours before they are required, the broth tubes being incubated at 37° C."

"*Mixing the serum with the broth culture.*—Three microscopic examinations have been made latterly, the serum in the mixtures being in the proportion of 50 per cent., 5 per cent., and 0·5 per cent. In order to do this a platinum loopful of the undiluted serum is mixed upon a clean coverslip, with the same loop full of the broth culture, thus making the 50 per cent. dilution. One loopful of the 10 per cent. serum, and of the 1 per cent. serum, mixed with a loopful of the broth culture, form a 5 per cent. and 0·5 per cent. dilution respectively. Immediately after mixing, the coverslips are inverted upon a hanging drop slide, and the time of mixing noted."

"*The reaction, and reaction time.*—When judging of a reaction it is necessary that practically the whole of the bacilli shall be grouped together, leaving only single bacilli free in the field. If

there are a few small clumps, but the majority of the bacilli are either motile or free in the field, I have called this a half reaction, ' $\frac{1}{2}+$.' As regards the time, I have always been very rigid, and have continued to adopt the time I suggested in 1897, viz., half-an-hour. If there is not a complete reaction within half-an-hour, even if it is complete within one or two hours, I have returned the result as ' $\frac{1}{2}+$,' that is 'no reaction.'"

I have taken a serum clumping in 5 per cent. strength in half-an-hour as being diagnostic of enteric fever as far as the reaction itself is concerned.

WIDAL'S REACTION.*

No. of Case.	Reaction.			Spleen felt.	Typhoid spots.	Results of Urine Examination : Organisms found.
	50 per cent.	5 per cent.	0.5 per cent.			
1 i.	—	—	—	Yes	Yes	
2 ii.	○	○	○	—	—	<i>Bacillus coli communis</i> : atypical bac. col. com.
3	+	+	+	Yes	Yes	<i>Staphylococci</i> .
4	+	+	○	No	No	<i>Staphylococci</i> .
5 iii.	—	+	—	Yes	Yes	<i>Staphylococci</i> .
6	+	+	$\frac{1}{2}+$	No	Yes	<i>Staphylococci</i> .
7	○	○	○	Yes	Yes	<i>Staphylococci</i> .
8	○	+	○	No	Yes	<i>Para-colon bacillus</i> . A bacillus resembling in many respects the bac. enteritidis of Gärtner. The typhoid bacillus. <i>Bac. coli</i> com. and <i>bacillus proteus vulgaris</i> .
9 i.	○	$\frac{1}{2}+$	$\frac{1}{2}+$	No	Yes	<i>Staphylococci</i> .
10 ii.	ppt. ○	+	$\frac{1}{2}+$	Yes	Yes	<i>Staphylococci</i> .
11	+	+	+	Yes	No	<i>Bacillus para-typhi abdominalis</i> . <i>Staphylococci</i> .
12 i.	○	○	○	Yes	Yes	<i>Bacillus typhi abdominalis</i> . <i>Staphylococci</i> . A proteus-like organism, etc.
12 ii.	○	$\frac{1}{2}+$	○			
13 i.	○	○	○	No	Yes	<i>Bacillus para-coli communis</i> .
14 ii.	○	+	○	Yes	Yes	Sterile (p.m.).
14 iv.	—	—	—			
15 i.	+	○	$\frac{1}{2}+$	Yes	Yes	<i>Staphylococci</i> (v.).
15 ii.	+	○	○			
15 iii.	ppt.	$\frac{1}{2}+$	○			
15 iv.	+	+	○			
15 v.	—	++	++			

NOTE i.—No Widal examination made in this case.

NOTE ii.—This serum, although being completely negative in all strengths to the bacillus typhi abdominalis, was found to react fully to a twenty-four hours' culture of

* I have to thank Mr. W. C. C. Pakes for his courtesy in allowing me to publish these Widal reactions.

the bacillus para-coli communis, isolated from the urine of the same case. Its reactions were 50 per cent. = +, 5 per cent. = +, 0.5 per cent. = 0.

NOTE iii.—In this case no Widal examination was made at Guy's Hospital, but a marked Widal reaction was reported from the Jenner Institute.

NOTE iv.—No Widal examination made.

NOTE v.—Finding that staphylococci were so often present, and bacillus typhi abdominalis much less frequently, it occurred to me that the growth of the one might inhibit the growth of the other. In order to find out if such were the case, I inoculated three broth tubes, one with the bacillus typhi abdominalis, one with staphylococcus, and one with a mixture of the two, and placed them under like conditions at 37° C. After seven days' growth, the bacilli were found to be chiefly in evidence.

Bacillus typhi abdominalis.—Broth, 37° twenty-four hours. Turbid, slight sediment. H.D., slightly motile bacilli.

G.F.B., 37° twenty-four hours. Turbid, no gas.

Staphylococcus.—Broth, 37° twenty-four hours. Good growth, turbid, slight sediment. H.D., cocci, chiefly in pairs.

G.F.B., 37° twenty-four hours. Turbid, no gas.

Bacillus typhi abdominalis + *Staphylococcus*.—Broth, 37°, twenty-four hours. Good growth, turbid, slight sediment.

G.F.B., 37° twenty-four hours. Turbid, no gas. H.D. (broth), bacilli very much more in evidence than cocci. H.D., seven days, bacilli chiefly in evidence.

N.B.—A serum clumping with 5 per cent. in thirty minutes (5 per cent. = +) is taken as alone being positive and diagnostic.

In Case 2 an interesting condition was observed. This case was one in which the patient had had a typical attack of enteric fever some eight months previously. The blood was not examined for the Widal reaction during the attack. Eight months after the attack, however, the serum, on being tested by Widal's method,

with a twenty-four hours' old culture of bacillus typhi abdominalis, was found to give a completely negative reaction in all dilutions. (50 per cent. = \circ , 5 per cent. = \circ , .5 per cent. = \circ). The urine of this patient contained at the time the bacillus paracoli communis. This had been grown in pure culture on gelatine. A broth tube was inoculated from the gelatine, and a twenty-four hours' old culture obtained. This was used with the serum instead of bacillus typhi abdominalis, and was found to give a positive result. 50 per cent. = +, 5 per cent. = +. (Gruber's reaction).

This condition of the blood is, I think, worthy of note, being negative when tested by Widal's method with the bacillus typhi abdominalis, but reacting well with the bacillus paracoli communis, isolated from the urine of the same case.

The question arises whether these cases which are on the borderland of enteric fever as regards their diagnosis, are really due to the bacillus typhi abdominalis. Mr. W. C. C. Pakes, in his article on Widal's reaction,⁴ in discussing the diagnosis, says, "Provisional diagnoses during the course of the disease have not been accepted, unless the physician has been satisfied, after the termination of the case, that it was really enterica or was really not enterica." This excludes a great number of cases, which may be said to be on the borderland of enteric fever. I should like to raise the question, whether some of the so-called aberrant forms of enteric fever, may not rather be due to a septicæmia, similar to enteric fever, but arising from an infection with bacilli, allied to the bacillus typhi abdominalis, or to some organism of the coli group. That an infection arising in the alimentary tract in this way, may give rise to an abdominal condition, closely simulating enteric fever.

In connection with this point, I would bring forward a remark by Dr. Rolleston, in an article on enteric cases, in the Imperial Yeomanry Hospital at Pretoria. He says,⁵ "It is noteworthy that a positive reaction was obtained in only 64.5 per cent. of cases clinically regarded as certainly enteric."

⁴ Widal's Reaction. By W. C. C. Pakes. Guy's Hosp. Rep. Vol. LV.

⁵ The Agglutination Reaction in Typhoid Fever. Memorandum by Dr. H. D. Rolleston. B.M.J., Oct. 12, 1901, p. 1084.

Mr. W. C. C. Pakes, however, gives a much higher percentage,⁶ as also does Gwynn.⁷ In one of the cases, in which I made a bacteriological examination of the heart-blood, kidney, and urine from post-mortem bladder, I found the bacillus para-coli communis in each in pure culture. This patient had died with the clinical symptoms of enteric fever. On post-mortem examination, the appearances were those seen in enteric fever. A positive Widal reaction had been obtained on one occasion.

This, I think, is suggestive of an organism distinct from the bacillus typhi abdominalis being concerned. I have not been able to go into the subject, as it is outside the scope of my thesis, but since my attention has been drawn to it in the course of my work, I have ventured to raise the point.

The subject is one of much difficulty, owing to the want of a proper classification of these allied groups of bacteria, and from the fact that the clinical phases which occur in the disease known as enteric fever, if not grouped together in such a way that "enteric fever" can be diagnosed, are described as "aberrant forms of enteric fever," or "abortive forms of enteric fever," or in some such way.

THE PATHOLOGY OF THE CONDITION.

The presence of the typhoid bacillus in the urine is difficult of explanation. It does not seem to appear in the urine early, for the earliest date on which I found the bacillus was on the twentieth day of the disease. On the other hand, it seems to continue until late in the disease, and in some cases the bacteriuria due to the typhoid bacillus or some allied organism continues far into convalescence (*vide* Case 2).

⁶ Guy's Hosp. Rep. Ibid.

⁷ "Positive results of Widal's Reaction in cases certainly typhoid 99.6 per cent. Of all cases 98.1 per cent." A Study of the Widal Reaction in 265 cases of Typhoid Fever. By Norman B. Gwynn, M.B. The Johns Hopkins Hospital Reports. Vol. viii.

I have been unable to find the bacillus typhi abdominalis in sections of the kidney, taken from post-mortem cases of enteric fever, that I have examined. Neither have I been able to cultivate the bacillus typhi abdominalis from the kidney on any media that I have used. I have, however, cultivated the bacillus para-coli communis from the kidney in one case, and in this case I also found the same organism in the urine taken from the post-mortem bladder. Both of these were in pure culture. On histological examination of the kidney, stained with carbol-thionin blue, I was, however, unable to detect the presence of any organisms in the substance of the kidney, either in the cortex or medulla. Unfortunately, in this case, no examination had been made of the urine during life, and the fact that the bacillus para-coli communis was found also in the heart-blood in pure culture rather lessens the value of this case, as the possibility of the bacilli getting into the blood after death has to be borne in mind.

That the condition is due to a stray bacillus getting into the bladder from the blood through the kidney and multiplying there has been suggested by Dr. Horton Smith,⁸ who discusses the question under the following heads:—

1. Filtration from the blood.—Dismiss this as there is extreme difficulty in finding bacilli in the blood during life, and the blood has been examined bacteriologically, when bacilli have been swarming in the urine, and no bacilli found.

2. Suppuration in kidney.—This is very rare, bacilluria comparatively common.

3. Entrance of a stray bacillus into bladder from blood, through the kidneys.—Dr. Horton Smith shews that of six specimens of urine taken and inoculated with typhoid bacilli, and incubated at 37° C., four shewed general turbidity from growth of the micro-organisms at the end of eighteen hours, while two did not. These latter, however, shewed good growth in forty-eight hours.

If, as he contends, 25 per cent. of patients suffer from bacilluria due to the typhoid bacillus in enteric fever, this explanation of the pathology seems hardly to meet the case. The possibility of some

⁸ Goulstonian Lectures on the Typhoid Bacillus and Typhoid Fever.—*Lancet*, 1900. Part I., p. 821, et seq.

morbid condition in the kidney, allowing the passage of the bacilli through it, is one to be considered.

The pathology of kidney disease is at present obscure, and many points in this connection require to be cleared up. Any case of damage to an organ, however slight, is sufficient to upset its physiological equilibrium. It has been shewn that the staphylococcus pyogenes aureus, injected into the circulation of a rabbit, in most cases, will produce no ill effects upon the healthy heart, but if the cardiac valves have been previously injured, an infective endocarditis will result.⁹ But it has been proved by Biedl and Kraus,¹⁰ that in some cases, bacilli may come through the *normal* kidney, for they showed that micro-organisms which circulate in the blood, can be excreted through the absolutely intact kidneys. To demonstrate this, they chloroformed dogs, fixed a sterilized cannula into the vena jugularis or femoralis, performed laparotomy on the animals, inserted cannulas into the ureters, and examined the urine thus obtained under all the necessary precautions, after having injected the staphylococcus pyogens aureus, the bacillus coli communis, and the bacillus anthracis into the veins. Cultures made from the urine, shewed the micro-organisms which had been injected; examination of the urine was negative as to blood or albumin. They concluded that the normal kidney, through its physiological function, is able to excrete the micro-organisms.

In discussing bacteriuria, Herman Goldenburg¹¹ says that the bacteria enter the bladder, either by *infection* or *auto-infection*. Quoting Ultzmann, he says, that bacteriuria is found in patients with malaria, and in those who work in dissecting rooms, where the infection takes place through the respiratory organs, and that he treated a patient with bacteriuria, complicated by hæmaturia of renal origin, general malaise, and emaciation due to malarial infection, rapid improvement following the administration of quinine and salol. Cases are more frequent in which bacteriuria is due to

⁹ Surgical Pathology and Morbid Anatomy. By Anthony A. Bowlby, F.R.C.S., 4th Edit., p. 49.

¹⁰ Quoted from Herman Goldenburg's article on Bacteriuria. Med. Rec., New York, 1896, p. 228.

¹¹ H. Goldenburg. Bacteriuria. Med. Record, New York, 1896, vol. 1., p. 228.

catheterisation with dirty catheters, and auto-infection takes place from the intestines, either directly through contiguity, or indirectly through absorption by the lymphatics. In connection with infection taking place through the respiratory organs, the following is instructive: Pneumococci were found in the urine in the case of a female patient, aged 60 years, who had an attack of croupous pneumonia. On the seventh day she had pain on micturition, and other symptoms of cystitis. On examination of the urine, Frænkel's pneumococcus was found.¹²

Wreden demonstrated in the laboratory of Professor Nencki, that after a slight artificial traumatism in the rectum of male rabbits, the bacillus coli communis could be found in the urine of the animals. He claimed from this, that bacteria enter the bladder directly through the lymphatics, which connect bladder and rectum. He demonstrated that in rabbits, after a superficial erosion of the rectal epithelium, fatty substances, such as oil and vaseline, which were introduced into the rectum, were found in the urine.

The theory that bacteria enter the bladder in this way seems to be highly probable. Morris¹³ states that "the lymphatics of the bladder pass partly backwards, beneath the peritoneum, to join the rectal lymphatics, or the lymphatics of the uterus and vagina in the female; and partly forward to join the prostatic lymphatics and the lymphatics of the vesiculæ seminales."

Sappey states,¹⁴ that the lymphatics of the bladder have not yet been fully worked out in man, although he has demonstrated their arrangement in the rabbit and dog.

That the lymphatics are enlarged during pregnancy is undoubtedly the case, and admitting that the bacilli gain admittance to the bladder through the lymphatics, one would expect them to get into the urine more easily, during pregnancy, or during the puerperal state.

¹² Pneumococci in the Urine. By G. Munro Smith. Bristol Med. Chir. Journ., 1895, vol. xiii., p. 115.

¹³ A Treatise on Human Anatomy, by various Authors. Edited by Henry Morris, M.A., M.B.

¹⁴ Description et Iconographie des Vaisseaux Lymphatiques Considérés chez L'Homme et les Vertébrés. Par Ph. C. Sappey, p. 124. Vaisseaux lymphatiques de la vessie.

This is borne out by Case 12, in which the patient was a woman aged 30 years, who two or three weeks before being attacked by enteric fever, had been delivered of a child. In her case the bacillus typhi abdominalis was found in the urine on the twentieth day of the disease, when the first examination was made. They were also present on the fifty-first day, when the last bacteriological examination of the urine was made. That they were present before the twentieth day is very probable.

GENERAL CONCLUSIONS OBTAINED FROM THE INVESTIGATION OF THE ABOVE CASES.

1. That there is no definite evidence that the typhoid bacillus comes through the kidney.
2. That sections of the kidney, appropriately stained, shewed no evidence of any bacilli.
3. That the entrance of the bacilli into the bladder through the lymphatics, is much more probable, and that Wreden's experiments are valuable in this direction.
4. That in the majority of cases of enteric fever the bacillus typhi abdominalis is not present in the urine.
5. That when it is present it occurs late rather than early in the disease.
6. That when it is present the urine is generally normal in other respects, or contains only a trace of albumin.
7. That other micro-organisms, such as the bacillus coli communis, and the atypical bacillus typhi abdominalis are sometimes present in the urine in cases of enteric fever, and during the period of convalescence.
8. That as a rule when the bacillus typhi abdominalis is present in the urine there are no local clinical symptoms.
9. That bacteriuria may continue far into convalescence.
10. That the action of urotropin is very effectual in freeing the urine from bacilli.

11. That the bacteriological examination of the urine may be useful in some cases for diagnosis.

12. That the patient should be warned as to the possibility of spreading infection by the urine during convalescence.*

In conclusion, I wish to offer my thanks to the physicians and assistant physicians for their kindness in allowing me to study these conditions of enteric fever, in their respective patients, in the wards of Guy's Hospital.

I wish also to thank Dr. Bryant and Dr. Fawcett for so generously giving me the opportunity of working at the morbid anatomy and bacteriology of the cases on which a post-mortem examination was made. And finally to express my appreciation of the kindness and courtesy of Mr. W. C. C. Pakes, in allowing me the use of the Bacteriological Laboratory, and giving me so many facilities for working, and to thank him for many valuable suggestions in technique.

* In the base hospitals in South Africa it is the rule for the officers of the Royal Army Medical Corps to retain enteric patients for seven weeks after the temperature has reached normal, on account of their spreading infection in this way.

Enteric Fever.

No.		ation een or	REMARKS.
ade ...	None ma	...	—
...	Five exa		In this case the patient was ...

LIST OF CASES.

ABBREVIATIONS.

B.T.A.	=	<i>Bacillus typhi abdominalis</i> .
B.C.C.	=	<i>Bacillus coli communis</i> .
H.D.	=	Hanging drop.
C.M.B.	=	Carbol-methylene blue.
C.F.	=	Carbol-fuchsin.
Gr.-W.	=	Gram-Weigert.
G.F.B.	=	Glucose-formate broth.
F.	=	Female.
M.	=	Male.

NOTE.—In every case when an organism has been obtained in pure culture, this organism has been worked out according to a definite scheme, similar reactions being carried out with each organism, and comparisons afterwards made.

CASE 1.—F., æt. 28 years. Admitted into Miriam Ward under Dr. Washbourn, on July 11th, 1901, for pain in abdomen.

Patient had been ill nine days before admission with severe headaches. She had had one shivering fit, and pain in abdomen. On admission, temperature 105°, pulse 134. A few scattered spots were present on the abdomen, the spleen was palpable, and rhonchi could be heard over both lungs. The motions were of a light yellow colour. The pulse was weak and feeble. Stimulants (strychnine, musk, digitalis, and champagne) were administered. The pulse, however, became more feeble, she became cyanosed and died seven days after admission from heart failure. The blood was not examined for Widal's reaction.

A post-mortem examination was made six and a-half hours after death. The right lung was cedematous. Three feet from the cæcum was a small ulcer perforating the mucous membrane of the small intestine, and affecting the muscular coat. Below this were numerous raised infiltrating Peyer's patches, in a few of which ulceration had occurred; in the majority, however, there was no ulceration. There was no perforation of the intestinal wall. The pancreas was tough; the spleen large and tough. Histological examination of the kidney shewed the absence of any organisms.

Bacteriological Examination of the Kidney.

Broth, 37° C. Eighteen hours; four days, nil.

Agar, 37° C. Eighteen hours; four days, nil.

G.F.B. (anaerobically) 37° C. Eighteen hours, nil; four days, nil.

Direct film. C.M.B. Nil.

Gr.-W. Nil.

The kidney was sterile.

CASE 2.—M., æt. 31 years. This case is one in which a definite attack of enteric fever, with pyrexia, severe headache, and extreme prostration and feeble pulse, and rose-red spots, occurred seven months before the first examination of the urine was made. No Widal's reaction was tried at the time,

but the test being performed nine months, after the temperature fell to normal, gave a negative reaction (50 per cent. = 0. 5 per cent. = 0. 0.5 per cent. = 0). When, however, the serum was examined, as in Gruber's reaction, using a broth culture made from a gelatine culture of an organism, obtained from the urine of the same case (the bacillus para-coli communis), it was found to cause clumping of the bacilli.

The notes recording this are as follows :—

Blood examination for Gruber's reaction—

50 per cent. commenced clumping in five minutes, fully clumped in twenty-five minutes.

50 per cent. slight signs of clumping in twelve minutes, fully clumped in thirty minutes.

0.5 per cent. not clumped in thirty minutes.

50 per cent. = +. 5 per cent. = +. 0.5 per cent. = 0.

There were no complications in this case. Five examinations of the urine were made. Four of these were made before, and one after the administration of urotropin. The quantity of urotropin given was sixty grains, in six doses of ten grains each, administered three times a day over a period of two days. After this the urine was found to be quite sterile. There was no albumin at any time. The first examination of the urine was made seven months after the temperature became normal, the final one three months after this.

SPECIMEN 1.—Urine.

Description.—A bacillus.

Motility.—In dextrose broth, after six hours' growth, very slightly motile.

Staining.—Stains with C.M.B. Decolorised by Gram-Weigert.

Broth (37° twenty-four hours).—Turbidity, white sediment; two days, very slight trace of indol.

Peptone water (37° twenty-four hours).—Slight turbidity.

Formate broth (37° twenty-four hours).—Much gas formation, slight turbidity.

Dextrose broth (37° twenty-four hours).—Much gas formation, turbidity.

Lactose broth (37° twenty-four hours).—Gas formation, turbidity.

Saccharose broth (37° twenty-four hours).—Much gas formation, turbidity.

Glycerine broth (37° twenty-four hours).—Much gas formation, turbidity.

Nitrate broth (37° twenty-four hours).—No gas; turbidity. Red colour with meta-phenylene diamine = nitrites.

Lead broth (37° twenty-four hours).—

Gelatin stab (20° twenty-four hours).—White growth down stab; no spreading on surface; gas formation; no liquefaction.

Gelatin streak (20° twenty-four hours).—Well marked, raised, moist-looking, white vigorous growth, no liquefaction.

Gelatin shake (20° twenty-four hours).—Slight turbidity, much gas, no liquefaction.

Gelatin plates (20° two days).—Moist looking, small, white colonies, no liquefaction.

Agar streak (37° twenty-four hours).—Well marked, raised moist looking growth, gas formation.

Agar plates (37° thirty-six hours).—Large white moist-looking growths; raised; large gas bubbles; very vigorous growth.

Blood-serum (37° twenty-four hours).—Vigorous, moist-looking, white, raised growth.

Litmus milk (37° twenty-four hours).—Acidity, milk entirely clotted.

Potato (37° twenty-four hours).—Raised, yellowish, dirty-looking, vigorous growth.

Anaerobic growth (37° twenty-four hours).—Gas, slight turbidity, slight sediment.

Laevulose peptone (twenty-four hours). Much gas formation. Turbidity.

= *Bacillus Coli Communis*.

SPECIMEN 2.—*Urine*.

Description.—A bacillus.

Motility.—Very slightly motile (two days agar).

Staining.—With C.M.B. Decolorised by Gram-Weigert.

Pleomorphism.—Long and short bacilli.

Broth (37° twenty-four hours).—Turbidity, flocculence, sediment; four days, do., no indol.

Peptone water (37° twenty-four hours).—Slight turbidity, flocculence, sediment; four days, do.; five days, no indol.

Formate broth (37° twenty-four hours).—Gas formation, slight turbidity; four days, slight scum, no gas.

Dextrose broth (37° twenty-four hours).—Gas formation, slight turbidity; four days, no gas, acid.

Lactose broth (37° twenty-four hours).—Gas formation, slight turbidity; four days, no gas, acid.

Saccharose broth (37° twenty-four hours).—Marked gas formation, slight turbidity; four days, no gas, acid, sediment.

Glycerine broth (37° twenty-four hours).—Gas formation, slight turbidity; four days, no gas, acid.

Nitrate broth (37° twenty-four hours).—Turbidity, white sediment; two days, red colour with metaphenylene diamine = Nitrites.

Lead broth (37° twenty-four hours).—Turbidity; four days, very slight H₂S.

Gelatin stab (20° twenty-four hours).—Whitish growth along stab, gas; four days, no liquefaction; twenty-five days, gas, no liquefaction.

Gelatin streak (20° twenty-four hours).—Raised, narrow, whitish growth; four days, increased, no liquefaction; twenty-five days, no liquefaction.

Gelatin shake (20° twenty-four hours).—Gas formation; four days, dotted colonies on surface, slight turbidity; twenty-five days, no liquefaction.

Agar streak (37° twenty-four hours).—Well-marked, raised, moist-looking growth, gas formation; four days, do.

Agar plates (37° three days).—Large white colonies; gas formation.

Blood-serum (37° twenty-four hours).—Spreading, white, dry, growth; four days, do.

Litmus milk (37° twenty-four hours).—Marked acidity, marked gas, some clotting; two days, marked clotting; four days, decolourised.

Potato (37° twenty-four hours).—Raised, white, dry growth; four days, raised, marked, yellowish-brown growth.

Anaerobic growth (37° twenty-four hours).—Gas formation; four days, gas formation.

Laevulose broth 37° (twenty-four hours).—Marked gas formation, turbidity; four days, no gas, sediment.

= Atypical *Bacillus Coli Communis* (atypical because of no indol formation).

SPECIMEN 3.—*Urine*. Acid, no albumen.

Description.—Bacilli, rather short and thick.

Motility.—? Slightly motile, agar. Definitely slightly motile (Dextrose broth six hours.)

Staining with carbol methylene blue. Decolorised by Gram-Weigert.

Broth (37° twenty-four hours).—Turbid, white sediment, flocculi, no gas; four days, trace of indol; five days, trace of indol.

Peptone water (37° twenty-four hours).—Turbid, white deposit; thirteen days, no indol.

Formate broth (37° twenty-four hours).—Turbid, flocculent deposit; four days, turbid, no gas, slight sediment.

Dextrose broth (37° two days).—Much gas, acid.

Lactose broth (37° twenty-four hours).—Turbid, acid, much gas; four days, no gas.

Saccharose broth (37° twenty-four hours).—Turbid, acid, plenty of gas; four days, turbid, acid, no gas.

Glycerine broth (37° twenty-four hours).—Turbid, acid, no gas; four days, turbid, acid, no gas.

Nitrate broth (37° twenty-four hours).—Turbid, sediment, no gas; four days, good nitrites (with metaphenylene diamine).

Lead broth (37° twenty-four hours).—Slight H_2S ; four days, good H_2S ; thirteen days, good H_2S .

Gelatin stab (20° twenty-four hours).—Small dotted colonies down stab, growth on surface, no liquefaction; thirteen days, no gas, no liquefaction.

Gelatin streak (20° twenty-four hours).—Good growth, raised, moist-looking white; thirteen days, no gas, no liquefaction.

Gelatin shake (20° twenty-four hours).—Good gas bubbles all through medium, slight turbidity; thirteen days, gas at bottom only.

Agar streak (37° twenty-four hours).—Good growth, raised, white, vigorous; gas formation in agar. Four days, increased.

Agar plates (37° twenty-four hours).—White colonies, gas; seven days, large, yellowish white, moist-looking colonies.

Blood-serum (37° twenty-four hours).—Raised, very thick, yellowish white growth; four days, increased; thirteen days, ditto.

Litmus milk (37° twenty-four hours).—Well-marked acidity, no clotting, scum; four days, completely clotted, decolorized; thirteen days, do.

Potato (37° twenty-four hours).—Raised, yellowish-white, moist-looking growth; four days, yellowish-brown, moist; thirteen days, very moist-looking, dirty-white growth.

Anærobic growth (37° twenty-four hours).—Turbid, good gas; four days, good gas.

= *Bacillus Coli Communis*.

SPECIMEN 4.—*Urine* (Acid. No albumin).

Description.—A short oval bacillus.

Motility.—Very slight motility (Dextrose broth).

Staining.—Faintly with C.M.B. Slightly with C.F. Decolourised by Gram-Weigert.

Pleomorphism.—Some larger forms.

Broth (37° twenty-four hours).—Turbid, white deposit, fair gas; two days, slight trace of gas; five days, no indol.

Formate broth (37° twenty-four hours).—Abundant gas; two days, alkaline, no gas; four days, alkaline.

Dextrose broth (37° twenty-four hours).—Abundant gas; two days, acid, no gas; four days, acid, no gas.

Lactose broth (37° twenty-four hours).—Abundant gas; two days, acid, no gas; four days, acid, no gas.

Saccharose broth (37° twenty-four hours).—Abundant gas; two days, acid, good gas; four days, acid, no gas.

Glycerine broth (37° twenty-four hours).—Very good gas; two days, acid, no gas; four days, acid, no gas.

Nitrate broth (37° twenty-four hours).—Abundant gas, good nitrites with metaphenylene diamine.

Lead broth (37° twenty-four hours).—Turbid, no H₂S; four days, good H₂S.

Gelatin stab (20° twenty-four hours).—Slight growth, no gas, no liquefaction.

Gelatin streak (20° twenty-four hours).—Fair growth, no gas, no liquefaction.

Gelatin shake (20° twenty-four hours).—Good gas bubbles, throughout medium; no liquefaction.

Agar streak (37° twenty-four hours).—Vigorous, white, raised, growth, some gas bubbles; two days, increased; four days, increased.

Agar plates (37° twenty-four hours).—White colonies; six days, large white colonies.

Blood-serum (37° twenty-four hours).—Dry, wrinkled-looking growth; four days, increased.

Litmus milk (37° twenty-four hours).—Acid, entirely clotted; two days, do. decolourised; four days, do.

Potato (37° twenty-four hours).—Dry, yellowish, raised dotted growth, over whole surface; four days, growth much raised.

Anærobic growth (37° twenty-four hours).—Turbid, abundant gas; two days, good gas; four days, good gas.

= Atypical *Bacillus Coli Communis*.

There is no Indol Reaction, differing thus from the typical *Bacillus Coli Communis*.

SPECIMEN 5.—*Urine*. No albumin. Acid.

Examined after the administration of six ten-grain doses of urotropin given three times a day for two days, and found to be sterile.

Broth 37° twenty-four hours, nil; two days, nil; four days, nil.

Glucose formate broth (anærobically) 37° twenty-four hours, nil; two days, nil; four days, nil.

CASE 3.—F. æt. 9 years. Admitted into Mary Ward under Dr. Taylor, on July 3rd, 1901, for headache and fever. Twelve days before admission she had a sore throat and headache. The throat improved but the headache continued, and she complained of pain in legs. She became very weak. The motions were watery and yellow. She has become thinner, and has great thirst. She has had fever five days. Condition on admission: Pulse 124, temperature 102·6°, respiration 28, urine 1024, acid; no albumin. Spleen not palpable. The temperature became normal on the fifth day after admission, and continued normal until the seventeenth, when it rose to 103·2°. (Interval=fourteen days). On the eighteenth day Widal's reaction was present. On the twenty-second, spots appeared on the abdomen, this being the

sixth day after the temperature rose; the spleen became palpable; on the twenty-third day more spots appeared. The temperature rose to 104·6°, and patient was sponged. On the twenty-fifth day, being the thirty-fifth day of the disease, including relapse interval, the urine was examined bacteriologically, and also on the thirty-ninth day of the disease.

July 28th. There are over thirty spots to-day.

July 30th. Some of the spots have faded, but there are still a large number. Bowels constipated. Glycerine enema.

August 1st. Most of the spots have faded. Temperature keeps below normal.

August 5th. Urine 1020, faintly acid. Albumin, blood and pus present.

Urotropin	gr. v.
Ammon. Beng....	gr. v.
Tinct. Hyoscyam.	℥ x.
Sp. Chlorof.	℥ v.
Inf. Uvæ Ursi ad	3 ss. bis die.

The urine unfortunately was not examined bacteriologically at this time.

August 8th. There are no spots. Temperature normal. The urine became normal after the exhibition of urotropin. Glycerine enema.

August 10th. Patient is going on well; temperature has not risen above 99° for nine days. Pulse 60, respiration 20. There are no spots.

August 12th. Condition is improving. The bowels are rather constipated. The urine is normal. Temperature 96·4°—98·6°. There are no spots.

August 21st. On full diet. Gets up after tea.

August 24th. Patient discharged.

July 12th. Widal reaction 50 per cent. = + 5 per cent. = + 0·5 per cent. = +.

SPECIMEN 1.

G.F.B. 37° (anærobically). Two days, nil.

Gelatine plate. Nil.

Two broth tubes added to flask at 37°. Twelve hours, nil; three days, nil.
= urine sterile.

SPECIMEN 2.

Hanging drop. Nil.

G.F.B., 37°. Twenty-four hours; turbidity.

Broth, 37°. Twenty-four hours; turbidity.

Agar plates, 37°. Twenty-four hours; a few white colonies.

C.M.B. Staphylococci only.

Gram-Weigert. Staphylococci present.

Staphylococci only.

CASE 4.—M., æt. 21 years. Admitted into Philip Ward under Dr. Taylor, on July 26th, for diarrhœa.

For three weeks before admission he suffered from headache. He did his work nevertheless. For seven days before admission patient was feeling very weak, and for three days had suffered with diarrhœa.

On admission, he was pale and drowsy, with signs of much weakness, with a foul tongue and breath. The abdomen was full, but the spleen could not be felt. There was an apical systolic bruit. There were no spots. The urine was normal. There was diarrhœa. The blood gave the Widal reaction,

The pyrexia continued for rather over three weeks and then dropped to normal, the patient being eventually discharged convalescent.

Three examinations of the urine were made, on each occasion staphylococci only were found.

July 27th. Widal's reaction, 50 per cent. = +. 5 per cent. = +. 0.5 per cent. = + 0.

SPECIMEN 1.—*Urine*. No albumin.

H.D. Urine, nil.

Broth, 37°. Twenty-four hours; cocci in pairs.

G.F.B., 27°. Twenty-four hours; no gas; slight turbidity (anærobically).

Agar plates, 37°. Twenty-four hours; large white colonies.

C.M.B. Staphylococci only.

Planted agar tube from agar plate. Twenty-four hours.

H.D. Cocci; C.M.B., staphylococci.

Gram-Weigert. Staphylococci (stain well).

Staphylococci only present.

SPECIMEN 2.

H.D. Urine, nil.

Broth, 37°. Twenty-four hours, turbid; no gas.

G.F.B., 37°. Twenty-four hours; no gas; cloudy; slight white sediment.

H.D., cocci only.

Agar plate, 37°. Twenty-four hours; a large number of large and small white colonies. G.M.B. Staphylococci only.

Staphylococci only present.

SPECIMEN 3.—*Urine*. No albumin.

H.D. Urine, nil.

Broth, 37°. Twenty-four hours; turbid; no gas; H.D., cocci only.

G.F.B., 37°. Twenty-four hours; slightly turbid; no gas.

Agar plate, 37°. Twenty-four hours; dotted white colonies.

C.M.B. Staphylococci only.

Staphylococci only present.

CASE 5.—F., æt. 32 years, married. Admitted into Miriam Ward under Dr. Washbourn, for continued fever and diarrhoea, on August 15th, 1901. Thirteen days before admission she was attacked with severe pains in the abdomen, and vomiting. On the day following diarrhoea set in, and has continued since. She has also suffered from headache, pain in the back and limbs, and weakness. On August 12th a specimen of her blood showed a marked Widal reaction when examined at the Jenner Institute. On admission: Temperature 103.2°, pulse 124, respiration 36. Tongue furred, face flushed, abdomen full, with a few faint rose-coloured spots. The spleen is not palpable. Pulse rapid, small, soft, regular. Heart-sounds normal. Many non-consonating râles to be heard in the chest. The pulse became rather feeble, and she was ordered brandy. On August 17th the spleen could be felt. The pulse is still feeble. On August 18th a few fresh spots were noticed on the abdomen. Diarrhoea continued, and Mist. Opii Acida was ordered.

The diarrhoea ceased, and patient gradually improved, and was discharged convalescent on September 27th. Bacteriological examinations of the urine were made on five occasions. On the twenty-fifth, twenty-seventh, thirty-fourth, forty-first and fifty-fifth day of the disease. Twice it was found to be quite sterile. On three occasions staphylococci only were found.

SPECIMEN 1.—Twenty-fifth day.

H.D., urine, nil.

Broth, 37° twenty-fours, nil. Four days, nil.

G.F.B. (anaerobic growth) 37° twenty-four hours, nil. Two days, nil. Four days, nil.

SPECIMEN 2.—Twenty-seventh day.

Broth, 37° twenty-four hours, nil. Turbidity.

Glucose formate broth 37° twenty-four hours, nil. Three days, turbidity.

Agar slope, twenty-four hours, nil.

C.M.B., staphylococci only present.

SPECIMEN 3.—Thirty-fourth day.

H.D., nil.

Broth, 37°, twenty-four hours, nil. Two days, white sediment, clear fluid.

G.F.B., 37°, twenty-four hours, nil. Two days, turbid.

Agar plate, 37°, twenty-four hours, nil. Two days, white colonies.

Planted agar plates from agar. Small round white and yellow colonies.

Staphylococci only.

SPECIMEN 4.—Forty-first day.

H.D., urine, nil.

Broth, 37°, twenty-four hours, nil. Two days, nil.

G.F.B., 37°, twenty-four hours, nil. Two days, nil.

Agar plates, twenty-four hours, nil.

The urine is sterile.

SPECIMEN 5.—Fifty-fifth day. Urine. No albumin.

H.D., urine, nil.

G.F.B., 37°, twenty-four hours. Turbid.

Broth, 37°, twenty-four hours. H.D. cocci.

Agar plate, 37°, twenty-four hours. White colonies. H.D. cocci.

Planted agar tube from agar plate. Twenty-four hours, very sticky growth of small white dotted colonies; vigorous growth. H.D. nil. C.M.B. nil. Planted a broth tube from agar tube as this so sticky that no organisms would adhere to cover slip. Planted gelatin stab 20°, three days. No liquefaction. C.M.B. cocci only. H.D. cocci only, chiefly in pairs. Planted broth 37°, seven days. H.D. cocci only.

CASE 6.—M., æt. 27 years. Admitted into Stephen Ward under care of Dr. Pitt, on August 15th, 1901, for pain in the head and abdomen. For three weeks previously he had suffered from pain in the head and the back of neck. He was weak, and suffered from giddiness.

On admission, temperature 103·2°, pulse 72. There is diarrhœa. There are two or three rose-coloured spots on abdomen which disappear on pressure. The tongue and breath are foul. The spleen cannot be felt. There are a few râles and rhonchi to be heard in the chest.

August 16th. More spots appearing.

August 17th. Temperature 103·6°. Patient gradually improved and was discharged convalescent on September 12th.

A bacteriological examination of the urine was made on two occasions. On each occasion staphylococci only were found.

August 17th. Widal reaction.

50 per cent. = + 5 per cent. = + 0·5 per cent. = + 4.

SPECIMEN 1.

H.D. Urine, nil.

Broth, 37°.

G.F.B., 37°. Twenty-four hours; H.D., cocci only, in short chains.

Planted agar from G.F.B. Twenty-four hours; small white and yellow colonies; cocci only.

Centrifugalized some (original) urine; planted broth; twenty-four hours; white sediment; staphylococci only.

Planted three gelatine plates from broth; twenty-four hours, nil; three days; staphylococci only.

Staphylococci only present.

SPECIMEN 2.

H.D. Urine, nil.

G.F.B., 27°. Twenty-four hours; slight turbidity; sediment; no gas; two days, do.

Broth, 37°. Twenty-four hours; turbidity; white sediment; two days, do.

Agar plate, 37°. Twenty-four hours; numerous small white colonies;

H.D., cocci.

Stained G.F.B. and agar plate growths with C.M.B. = staphylococci only.

CASE 7.—M., æt. 29 years. Admitted into John Ward under Dr. Washbourn on August 15th, 1901, for pyrexia and albuminuria.

Seven weeks ago he had severe diarrhoea, which lasted about a month. The motions were green. He had also slight headache. He was kept on milk and soda water for a fortnight, and after that on custards. He was told that he had gastritis. He remained in bed for three weeks, and, after that he did no work for three weeks as he felt very weak. A week ago he caught a chill and went to see his doctor, who took his temperature and sent him to bed. He was told he had a relapse, and he was sent to Guy's Hospital after having been in bed again for a week, during which he felt weak, but was otherwise feeling well.

On admission, the pulse was 108, temperature 100·2°. A few raised rose-red spots which fade on pressure are present on the abdomen. The heart is normal. Râles and rhonchi can be heard in the chest. The abdomen is not distended. The spleen is palpable. The pharynx is injected and sore. Tonsils swollen and covered with mucus. No diarrhoea. Urine 1015; Neutral; albumin, phosphates, and urates present. The blood was negative to the Widal reaction.

August 16th. The spots have disappeared. No suspicious new ones have appeared. Stools liquid, brown, not offensive. Diet, milk and soda water, brandy.

August 17th. A small quantity of blood in motions this morning.

August 18th. Temperature 103°, sponged.

August 19th. Pulse 120, regular, weak. No definite spots. Spleen palpable.

August 23. Sponging does not reduce temperature. Quinine, grs. xx. This had apparently a marked effect. A trace of blood in motions. The blood is still negative to Widal's reaction.

August 26th. Temperature 104°. Great hæmorrhages from bowels this morning, and he was much paler. Morphia was administered, and later lead

and opium, and tannic acid, ice compresses to abdomen. These had, however, no effect, and patient died.

Post-mortem examination.—Spleen much enlarged. Ulceration of small and large intestine. Iron found free in liver.

August 16th. Widal reaction.

50 per cent. = 0. 5 per cent. = 0. 0·5 per cent. = 0.

Spleen.

Description.—Short bacilli.

Motility.—Fair motility.

Staining.—Fairly well with C.M.B. Decolorised by Gram-Weigert.

Broth.—(37° twenty-four hours).—Turbidity, no gas; four days, no indol.

Peptone water (37° twenty-four hours).—Turbidity, no gas; four days, do.; five days, no indol.

Formate broth (37° twenty-four hours).—No gas; four days, no gas.

Dextrose broth (37° twenty-four hours).—Acid, turbid, no gas; four days, do.

Lactose broth (37° twenty-four hours).—No gas; four days, do.

Saccharose broth (37° twenty-four hours).—No gas; four days, do.

Glycerine broth (37° twenty-four hours).—No gas; four days, do.

Nitrate broth (37° twenty-four hours).—Turbid, sediment, no gas; two days, abundant nitrites (with metaphenylene diamine).

Lead broth (37° twenty-four hours).—Turbidity, slight H₂S.; four days, do.

Gelatin stab (20° twenty-four hours).—Growth along stab; four days, do., some slightly spreading growth on surface.

Gelatin streak (20° twenty-four hours).—Slight greyish white growth; four days, do.

Gelatin shake (20° twenty-four hours).—Turbid, no gas; four days, no gas.

Agar streak (37° twenty-four hours).—Slight, white growth; four days, increased slightly but not vigorous.

Blood-serum (37° twenty-four hours).—Slight white growth; four days, do.

Litmus milk (37° twenty-four hours).—Nil; two days, distinctly acid, no clotting; four days do., no clotting.

Potato (37° twenty-four hours).—Moist-looking growth; four days, do., a transparent film.

Anaerobic growth (37° twenty-four hours).—Turbid, no gas; four days, no gas, slightly turbid.

Gruber's reaction.—50 per cent. = +. 5 per cent. = ++. 0·5 per cent. = +.

NOTE.—Experiment done with a twenty-four hours' broth culture grown from gelatine, using a serum that had previously reacted perfectly with a known bacillus typhi abdominalis.

= *Bacillus Typhi Abdominalis*.

Bacteriological examination of urine from post-mortem bladder.

In the examination of the urine from the post-mortem bladder, the surface of the bladder was sterilized with a searing iron, and an incision made with a sterile knife through the bladder wall. Some urine was now drawn up by means of a sterilized pipette, and transferred to a sterilized flask.

Broth, 37°. Twenty-four hours; turbid; flocculent.

G.F.B., 37°. Twenty-four hours; turbid; flocculent.

Agar, 37°. Twenty-four hours; small, white, colonies dotted over the surface.

Staphylococci only were found in these.

Bacteriological examination of kidney.

Agar, sloped, 37°. Twenty-four hours, nil; two days, nil; four days, nil.

Broth, 37°. Twenty-four hours, nil; two days, nil; four days, nil.

G.F.B., 37°. Twenty-four hours, nil; two days, nil; four days, nil.

The kidney is sterile.

Histological examination of a section of the kidney stained with carbol thionin blue shewed the absence of any organisms.

CASE 8.—F., æt. 3 years and 10 months. Admitted under the care of Dr. Taylor, into Mary Ward, on August 26th, for diarrhœa. Four days ago she was taken ill with diarrhœa. There has been no vomiting. She has been drowsy, and has not appeared to be in pain. On admission, temperature 103°, pulse, 120, respiration 32. Heart-sounds normal. Lungs normal. The abdomen is full and there are some raised spots upon it. There is diarrhœa.

August 27th. Temperature 104.4°. More spots have appeared on the abdomen.

August 29th. Temperature 103.6. The blood gives a well-marked Widal reaction. 50 per cent. = O. 5 per cent. = +. 0.5 per cent. = O.

September 2nd. Temperature 104°. Abdomen tense and rather full.

September 5th. Temperature 102.8°. Diarrhœa still continues. Abdomen less distended. Spleen not palpable.

September 8th. Diarrhœa less.

September 14th. Diarrhœa almost disappeared. Temperature 101.4°.

September 18th. Patient much improved. Spleen not palpable.

October 1st. Bowels constipated. Enemata. Temperature 99°

October 3rd. Farinaceous diet.

Bacteriological examinations of the urine were made on five occasions, on the eighth, thirteenth, twentieth, thirtieth, and forty-seventh days of the disease respectively. The first examination shewed an atypical colon bacillus to be present in pure culture. The second examination shewed the presence in pure culture of a bacillus, similar to the bacillus enteritidis of Gärtner, but differing from it in that no alkalinity of milk was produced. The third examination shewed the presence of the bacillus typhi abdominalis in pure culture. In the fourth examination staphylococci were found, and a bacillus which was probably proteus vulgaris, as it caused liquefaction of gelatine among other reactions, thus differentiating it at once from the typhoid and coli groups. The fifth examination shewed the presence of the bacillus coli communis and the bacillus proteus vulgaris.

SPECIMEN 1.—Urine. No albumin. Catheterized, with aseptic precautions.

Description.—A bacillus.

Motility.—Fair motility.

Staining with C.M.B. Decolorised by Gram-Weigert.

Broth (37° twenty-four hours).—Turbidity, no gas; two days, turbidity, sediment, no gas; six days, good indol.

Peptone water (37° twenty-four hours).—Turbidity; six days, good indol.

Formate broth (37° twenty-four hours).—Marked gas formation; two days, no gas, no colour change; six days do.

Dextrose broth (37° twenty-four hours).—Good gas formation, acid; two days, no gas, acid; six days, do.

Lactose broth (37° twenty-four hours).—Good gas formation, acid; two days, no gas; six days, acid.

Saccharose broth (37° twenty-four hours).—No gas, no colour change, slightly turbid.

Glycerine broth (37° twenty-four hours).—Nil; two days, good gas formation, slightly acid; six days, no gas, acid.

Nitrate broth (37° twenty-four hours).—Marked turbidity, gas, good nitrites (with metaphenylene-diamine).

Lead broth (37° twenty-four hours).—Marked turbidity, no H_2S ; two days, slight H_2S ; six days, do.

Gelatin stab (20° twenty-four hours).—Slight growth along stab, no gas; two days do.; six days, no liquefaction, growth on surface.

Gelatin streak (20° twenty-four hours).—Slight growth; two days, transparent growth, irregular edges, no liquefaction; six days do.

Gelatin shake (20° twenty-four hours).—Turbidity, some gas bubbles, no liquefaction; two days do.; six days do., cloudy.

Agar streak (37° twenty-four hours).—Vigorous, spreading greyish white growth; six days do.

Blood-serum (37° twenty-four hours).—Well marked, raised white growth; two days do.; six days do.

Litmus milk (37° twenty-four hours).—Marked acidity, no clotting; six days, no clotting; twelve days, no clotting.

Potato (37° twenty-four hours).—Very slight whitish growth; two days do.; six days, raised yellowish white growth.

Anaerobic growth (37° twenty-four hours).—Turbidity, marked gas production.

= Atypical bacillus coli communis.

Differs from bacillus coli communis in that it does not clot milk.

SPECIMEN 2.—Urine.

Description.—A bacillus.

Motility.—Very motile.

Staining.—Stains fairly with C.M.B. Decolorised by Gram-Weigert.

Pleomorphism.—Longer and shorter forms.

Broth (37° twenty-four hours).—Marked turbidity, white deposit on sides, sediment, no gas; two days, do.; four days, no indol.

Peptone water (37° twenty-four hours).—Slight turbidity, sediment, no gas; two days, do.; four days, do.; ten days, no indol.

Formate broth (37° twenty-four hours).—Slight turbidity, sediment, marked gas formation, no acidity; two days, no gas, alkalinity; four days, do.

Dextrose broth (37° twenty-four hours).—Sediment, acidity, marked gas formation; two days, no gas; four days, do.

Lactose broth (37° twenty-four hours).—Sediment, acidity, gas formation; two days, no gas; four days, do.

Saccharose broth (37° twenty-four hours).—No gas, no change; two days, slight turbidity; four days, do.

Glycerine broth (37° twenty-four hours).—Distinctly acid, no gas; two days, do.; four days, do.

Nitrate broth (37° twenty-four hours).—Turbidity, sediment, no gas; two days, do., good nitrites with metaphenylene diamine.

Lead broth (37° twenty-four hours).—Turbidity, very slight H_2S ; two days, fair H_2S ; four days, good H_2S .

Gelatin stab (20° twenty-four hours).—Slight growth along stab none on surface; four days' growth spreading on surface.

Gelatin streak (20° twenty-four hours).—Semi-translucent growth with irregular edge, no liquefaction; two days, do.; four days, good growth.

Gelatin shake (20° twenty-four hours).—Slight turbidity, good gas bubbles, no liquefaction; two days, do.; note that gas bubbles are not within half an inch of surface; four days, growth on surface.

Agar streak (37° twenty-four hours).—Vigorous, yellowish white growth; two days, do.; four days, spreading growth on surface.

Agar plates (37° twenty-four hours).—Whitish colonies.

Blood-serum (37° twenty-four hours).—Raised, yellowish white growth two days, do.; four days, do.

Litmus milk (37° twenty-four hours).—Marked acidity, no clotting; four days, no clotting; ten days, no clotting; thirty days, no clotting.

Potato (37° twenty-four hours).—Very slight growth; two days, do.; four days, thick raised growth.

Anærobic growth (37° twenty-four hours).—Turbidity, very marked gas formation; two days, do.; four days, still some gas; ten days, no gas, sediment.

This bacillus is similar to the bacillus enteritidis of Gärtner but differs from it in that no alkalinity of milk is produced.

SPECIMEN 3.—Urine.

Description.—A short bacillus, slightly motile (agar).

Staining.—Stains faintly with C.M.B., well with C.F., decolorised with Gram-Weigert.

Pleomorphism.—Longer and shorter forms.

Broth (37° twenty-four hours).—Slightly turbid, no gas; three days do; four days, no indol.

Peptone water (37° twenty-four hours).—No gas; three days, nil; six days, no indol.

Formate broth (37° twenty-four hours).—Very slightly turbid, no gas; three days, no gas, slightly alkaline; eight days do.

Dextrose broth (37° twenty-four hours).—Acid, no gas, slightly turbid three days do.

Lactose broth (37° twenty-four hours).—Nil; two days, slightly red, no gas; six days, acid, no gas.

Saccharose broth (37° twenty-four hours).—Nil, no gas; three days, nil six days, nil.

Glycerine broth (37° twenty-four hours).—No gas; three days, nil; six days, nil.

Nitrate broth (37° twenty-four hours).—Slight nitrites (with metaphenylene diamine).

Lead broth (37° twenty-four hours).—No H_2S ; three days, no H_2S ; six days, no H_2S ; thirteen days, no H_2S ; twenty-one days, no H_2S .

Gelatin stab (20° twenty-four hours).—Nil, no gas; six days, very slight growth down stab; ten days, no gas, no liquefaction.

Gelatin streak (20° twenty-four hours).—Very slight growth; six days do., no gas; ten days, no gas; no liquefaction.

Gelatin shake (37° twenty-four hours).—No gas, nil; six days, nil, no gas; ten days, no gas, no liquefaction.

Agar streak (37° twenty-four hours).—Slight white growth, dotted colonies; six days, do.

Blood-serum (37° twenty-four hours).—Very slight growth, dotted colonies; three days, do; six days, do.

Litmus milk (37° twenty-four hours).—Acid, no clotting; six days, do.; eight days, no clotting; twenty-one days, no clotting.

Potato (37° twenty-four hours).—Nil; six days, nil; eight days, very slight growth.

Anærobic growth (37° twenty-four hours).—Slight turbidity, no gas; three days, do; six days, clear, sediment.

= *Bacillus typhi abdominalis*.

SPECIMEN 4.—*Urine*. No albumin.

20th September, 1901. H.D. *Urine*, nil.

Glucose formate broth, 37°. Twenty-four hours; turbid, gas, white flocculi.

Broth, 37°. Twenty-four hours; turbid, no gas, flocculi.

Agar plate, 37°. Twenty-four hours; a few white colonies, and an uniform growth spreading over plate.

23rd September, 1901. C.M.B. *agar*. Staphylococci only.

C.F. *broth*. Bacilli, many well stained. Made three *agar* plates from *broth*.

24th September, 1901. Rather short bacilli, very motile, stained with C.M.B.

Planted *agar tube*, 37°. Twenty-four hours; growth simulates that of *proteus*.

Formate broth, 37°. Gas, slight acidity.

Gelatin slope, 20°. Twenty-four hours; pure culture; liquefaction of *gelatin*; this excludes the *coli* and *typhoid* groups. Probably *proteus vulgaris*. The organism was not further worked out.

SPECIMEN 5.—*Urine*.

Description.—Short thick bacilli.

Motility.—Slightly motile.

Staining.—Fairly stained with C.M.B., well stained with C.F., decolorised with Gram-Weigert.

Pleomorphism.—Short oval bacilli, some longer forms.

Broth (37° twenty-four hours).—Turbid, sediment, no gas; three days, do.; five days, good indol.

Peptone water (37° twenty-four hours).—Turbid, no gas; two days, turbid, no gas.

Formate broth (37° twenty-four hours).—Turbid, gas formation, not acid; two days, no gas; four days, turbid, alkaline; no gas.

Dextrose broth (37° six hours).—Acid, turbid, abundant gas; twenty-four hours, no gas.

Lactose broth (37° twenty-four hours).—Acid, gas; two days, acid, no gas; three days, acid, fair gas; four days, acid, no gas.

Saccharose broth (37° twenty-four hours).—Slightly acid, no gas; two days, acid, very slight gas; three days, acid, very slight gas; four days, do.

Glycerine broth (37° twenty-four hours).—Turbid, no gas; two days, fair gas; three days, good gas; four days, acid, good gas.

Nitrate broth (37° twenty-four hours).—Turbid, good nitrites (with *meta-phenylene diamine*).

Lead broth (37° twenty-four hours).—Turbid, no H₂S; five days, good H₂S.

Gelatin stab (20° twenty-four hours).—Slight growth down stab, some spreading on surface, no gas, no liquefaction; nine days, do.

Gelatin streak (20° twenty-four hours).—Some white growth, no gas, no liquefaction; three days, no gas, no liquefaction; nine days do.

Gelatin shake (20° twenty-four hours).—Three days, turbid, no gas, no liquefaction; nine days do.

Agar streak (37° two days).—Vigorous, raised, white growth.

Agar plates (37° two days).—White colonies.

Blood-serum (37° twenty-four hours).—Raised, whitish growth; two days, increased.

Litmus milk (37° twenty-four hours).—Marked acidity, milk clotted; two days, marked acidity, milk entirely clotted; three days, do.

Potato (37° twenty-four hours).—Moist brownish growth; two days, moist brownish growth; four days, do.

Anaerobic growth (37° twenty-four hours).—Very abundant gas, turbid; two days, turbid, gas; three days, turbid, no gas.

= *Bacillus coli communis*.

Gruber's reaction.—This reaction was carried out with a twenty-four hours' old broth culture grown from gelatin. The serum used gave the following result when used with B.T.A. for Widal's reaction :—

5 per cent. = + +. 0·5 per cent. = + +.

On carrying out Gruber's reaction with the above organism, a completely negative reaction was given. Thus :—

50 per cent. = ○. 5 per cent. = ○. 0·5 per cent. = ○.

SPECIMEN 5 (2).

Gelatin stab (twenty-four hours).—Proteus-like growth; two days, liquefaction of gelatine.

Formate broth (twenty-four hours).—Good gas; four days, alkaline.

Litmus milk (twenty-four hours).—Acid; no clotting; three days, precipitation of casein.

Agar (twenty-four hours).—Spreading; greyish white growth.

= *Bacillus proteus vulgaris*.

CASE 9.—M., æt. 16 years. Admitted into John Ward under Dr. Shaw, on August 31st, for pyrexia and headache.

For a week previous to admission he had felt weak and giddy, and had suffered from headache. He had kept at his work until three days before admission. No epistaxis. No diarrhoea.

On admission. The abdomen was not distended, the spleen was not palpable. There was no diarrhoea, the bowels being rather inclined to be constipated. Pulse 120. Heart and lungs normal. Temperature 103·2°.

September 3rd. A partial Widal reaction was obtained. 50 per cent. = —. 5 per cent. = +₂. 5 per cent. = +₄.

September 5th. A complete Widal reaction was obtained. 50 per cent. = ppt. 5 per cent. = +. 5 per cent. = +₂.

The temperature fell to normal at the end of the second week, and patient made an uninterrupted recovery.

A bacteriological examination of the urine was made on the thirteenth, eighteenth, twenty-first, twenty-fourth, twenty-seventh, and fortieth day of the disease.

On each occasion cocci only were found, these in most instances being staphylococci.

SPECIMEN 1.

H.D. Urine, nil.

Broth, 37°. Twenty-four hours; white sediment; white growth along sides of tube.

G.F.B., 37°. Twenty-four hours; no gas; slight white sediment. H.D., cocci.

Agar plate, 37°. Twenty-four hours; white colonies; cocci only, chiefly in groups.

C.M.B. Staphylococci.

Staphylococci only present.

SPECIMEN 2.—Eighteenth day.

September 10th, 1901. H.D., nil.

G.F.B., 37°. Twenty-four hours, nil; two days, turbid; no gas; white sediment.

Broth, 37°. Twenty-four hours, nil; two days, turbid; no gas; white sediment.

Agar plate, 37°. Twenty-four hours, nil; white colonies.

September 12th, 1901. G.F.B. H.D., cocci.

Agar plate, C.M.B. Staphylococci only.

Staphylococci only.

SPECIMEN 3.—Twenty-first day. Urine. No albumin.

September 13th, 1901. H.D. Urine, nil.

G.F.B., 37°. Twenty-four hours, turbid; white deposit on sides and bottom of tube.

Broth, 37°. Twenty-four hours, turbid; white deposit on sides and bottom of tube. H.D., cocci.

Cocci in short chains with C.F.

Planted agar tube. Twenty-four hours. H.D., cocci.

C.M.B. Staphylococci.

Staphylococci only.

SPECIMEN 4.—Twenty-fourth day of disease. Urine, no albumin.

September 16th, 1901. H.D. Nil.

G.F.B., 37°. Twenty-four hours, nil; two days, turbid; no gas; cocci.

Broth, 37°. Twenty-four hours, nil; two days, turbid; no gas.

Agar plate, 37°. Twenty-four hours, nil; two days, white colonies.

C.M.B. from agar plate. Staphylococci only.

Staphylococci only.

SPECIMEN 5.—Twenty-seventh day of disease.

September 19th, 1901. H.D. Urine, nil.

G.F.B., 37°. Twenty-four hours, nil; two days, turbid; slight gas.

Broth, 37°. Twenty-four hours, nil; two days, turbid; no gas.

September 20th. Agar plate, 37°. Twenty-four hours; a few white colonies.

H.D. Broth; cocci only.

C.M.B. Staphylococci only.

Staphylococci only present.

SPECIMEN 6.—Fortieth day of disease. *Urine*.—Slightly acid; excess of phosphates; no albumin.

October 2nd, 1901. G.F.B., 37°. Twenty-four hours, clear; no gas. H.D., Cocci chiefly in pairs.

Broth, 37°. Twenty-four hours. Slightly turbid; no gas.

C.M.B. Cocci only.

H.D. Cocci only, chiefly in pairs.

Agar, 37°. Twenty-four hours, no growth; thirty-six hours, nil.

October 4th. G.F.B. H.D., repeated; cocci only, chiefly in pairs.

C.M.B. Cocci only.

CASE 10. M., æt. 11 years. Admitted into Philip Ward under the care of Dr. Taylor, on August 28th, 1901, for headache, weakness, and pain in right groin. He was affected with headache for nine days before admission.

On admission, temperature 100·2°, pulse 88, respiration 24. He is pale and drowsy and lies flat on his back. Tongue clean in middle but furred at edges. There is pain in the left groin, where the glands are enlarged. The bowels are regular, there is no abdominal tenderness. The spleen is palpable. A few rose-coloured spots are present on the right side of the abdomen. Respiratory system normal. Circulatory system normal. *Urine* 1010, neutral, otherwise normal.

August 30th. Patient feels well; spleen palpable.

September 10th. Temperature falling every day; the spleen is not palpable; no spots have been seen for a week.

September 16th. Temperature 103°, pulse 132, respiration 24. He had pain in his abdomen yesterday, and again this afternoon.

September 17th. Pain in the abdomen immediately after taking milk. There is no distension, and the abdomen moves well with respiration. The recti contract well and without pain when patient moves.

September 21st. There is no abdominal pain; no distension.

September 24th. Patient improving, temperature 100°.

October 2nd. Temperature 99·4°; spleen not palpable. *Urine* 1014, neutral, no albumin or sugar, no sediment, a little mucus.

October 4th. Temperature 99°.

October 7th. Farinaceous diet.

October 12th. Full diet.

The urine was examined bacteriologically on five occasions. On each of these staphylococci only were found. The examinations were made on the twenty-fifth, twenty-ninth, thirty-ninth, forty-seventh, and fifty-eighth day of the disease.

October 16th. Widal reaction.

50 per cent. = ○. 5 per cent. = ○. 0·5 per cent. = ○.

SPECIMEN 1.

12th September, 1901. H.D. *Urine*, nil.

G.F.B., 37°. Twenty-four hours; turbid; no gas; white deposit; cocci C.F.

Broth, 37°. Twenty-four hours; slight white sediment; no turbidity.

Agar plate, 37°. Twenty-four hours; small white colonies; H.D., Staphylococci.

13th September. Planted agar plates from G.F.B. Two days, small, round, raised white colonies on plates; H.D., cocci only.

C.M.B. Staphylococci only.

SPECIMEN 2.

16th September, 1901. H.D. Urine, nil.

G.F.B., 37°. Twenty-four hours; very turbid; very slight gas; H.D., cocci.

Broth, 37°. Twenty-four hours; very small white dotted colonies.

Agar plate, 37°. Twenty-four hours; small white colonies.

C.M.B. Staphylococci only.

C.F. Staphylococci only.

SPECIMEN 3.

26th September, 1901. H.D. Urine, nil.

G.F.B., 37°. Twenty-four hours; very turbid; no gas; white sediment.

H.D. Cocci only, in groups.

C.M.B. Staphylococci only.

Broth, 37°. Twenty-four hours; turbid; no gas.

Agar plate. Twenty-four hours; white colonies.

C.M.B. Staphylococci only.

SPECIMEN 4.

Urine.—Neutral, cloudy; no albumin; excess phosphates.

5th October, 1901. H.D. Urine, nil.

G.F.B., 37°. Twenty-four hours; turbid; flocculent deposit; no gas;

H.D., cocci only; C.M.B., *staphylococci*.

Broth, 37°. Twenty-four hours; turbid; yellowish white deposit; no gas

H.D., cocci only, in pairs and masses.

Agar plate, 37°. Twenty-four hours; small colonies; H.D., cocci only.

Staphylococci only.

SPECIMEN 5. Urine.—Acid, no albumin; excess phosphates.

15th October, 1901. H.D. Urine, nil.

G.F.B., 37°. Twenty-four hours; turbid; no gas; H.D., cocci only in groups; C.M.B., *staphylococci* only.

Broth, 37°. Twenty-four hours; turbid; no gas; H.D., cocci only; C.M.B. Staphylococci only.

Agar, 37°. Twenty-four hours; small white colonies; two days, increased; H.D., cocci only, in groups.

Staphylococci only.

CASE 11.—M., æt. 25 years. Admitted on September 11th, 1901, into Stephen Ward under care of Dr. Bryant, for diarrhœa.

One month previous to admission he complained of general lassitude, headache, diarrhœa, and pain in right side of abdomen. He vomited once. The diarrhœa continued in spite of treatment; the number of stools was six or eight a day, in character they were liquid and light yellow. He quite lost his appetite and has been taking milk only. He has been growing steadily weaker and has lost flesh considerably.

On admission, temperature 98°, pulse 72, respiration 24. Patient is thin, but the abdomen is moderately full. The spleen is palpable; the diarrhœa is continual. The abdomen is not generally tender, but there is tenderness in one spot in the left iliac region. There are no spots. The tongue is clean in middle but furred at the sides. The breath is not foul. There is a rough

systolic bruit over the mitral area. He has a slight cough. The urine is normal.

September 17. The blood gives a good Widal reaction. He is much better, the diarrhoea is less, temperature is normal.

September 20th. Temperature 100·2°.

September 22nd. Temperature 102·2°.

September 26th. He is suffering from a relapse; the temperature is keeping up. The diarrhoea, however, has ceased.

September 27th. There is still a little fulness of the abdomen, which is slightly tender on pressure, especially on the right side.

October 3rd. He is constipated but otherwise is going on very well. Temperature 101°.

October 6th. Temperature 101°. He feels well; there are no spots.

October 9th. Temperature 99°. There is still constipation.

October 10th. Patient is going on very well.

September 17th, 1901.

50 per cent. = +. 5 per cent. = +. ·5 per cent. = +.

A bacteriological examination of the urine was made on three occasions. On the first occasion staphylococci and para-typhi abdominalis bacilli were found.

On the second occasion staphylococci only were found. On the third, staphylococci and a bacillus approaching the bacillus proteus vulgaris in reactions.

SPECIMEN 1.—*Urine*, normal.

H.D. Urine, nil.

G.F.B., 37°. Eighteen hours; turbid; no gas; H.D., cocci in short chains; bacilli, slightly motile.

Broth, 37°. Eighteen hours; turbid, flocculent growth on sides of tube; no gas.

Agar plate, 37°. Eighteen hours; white colonies; staphylococci only.

September 18th, 1901. Made three agar plates; twenty-four hours; white colonies.

September 19th. H.D. cocci; C.M.G., short bacilli; C.F., do.

Planted agar tube and glucose formate broth (anaerobically) from agar plate.

September 20th. Short bacilli and cocci in chains of three or four in glucose formate broth.

Short oval bacilli in pure culture on agar; two small white colonies. Staphylococci found plus bacilli.

These bacilli were planted on agar, and finally worked fully out as given hereunder.

SPECIMEN 1.—*Urine* (normal).

Description.—Short oval bacilli, thick, many in pairs jointed together.

Motility.—Slightly motile (agar), slightly motile (dextrose broth six hours).

Staining.—Stain well with C.M.B., faintly with C.F., decolorised by Gram-Weigert.

Broth (37° twenty-four hours).—Turbid, no gas; three days, do, white sediment; five days, no indol.

Peptone water (37° twenty-four hours).—Nil; three days, nil; five days, scum on surface, slight turbidity; twenty-one days, no indol.

Formate broth (37° twenty-four hours).—Slight turbidity, no gas, deposit, bleaching, slight reddening; three days, reddened, turbid, no gas; five days, do.

Dextrose broth (37° twenty-four hours).—Two days, acid, no gas.

Lactose broth (37° twenty-four hours).—Turbid, no gas, bleaching; three days, gas, acid, turbid; five days, do., no gas.

Saccharose broth (37° twenty-four hours).—Slightly turbid, no gas; three days, acid, no gas; five days, do.

Glycerine broth (37° twenty-four hours).—Slightly turbid, no gas, bleaching, streaks of blue; three days, almost colourless; five days, do.

Nitrate broth (37° twenty-four hours).—Turbid, no gas, no nitrites (with metaphenylene-diamine).

Iron or lead broth (37° twenty-four hours).—Two days, good H₂S.

Gelatin stab (20° twenty-four hours).—Very slight growth, no liquefaction; three days, increased; no surface growth; five days, no gas; no liquefaction.

Gelatin streak (20° twenty-four hours).—Very slight growth, no liquefaction; five days, no gas; no liquefaction.

Gelatin shake (20° twenty-four hours).—Streaked turbidity, no gas, no liquefaction; five days, no gas, no liquefaction.

Agar streak (37° twenty-four hours).—Slight, white, spreading, growth; three days, increased; five days, do.

Blood-serum (37° twenty-four hours).—Very slight growth; three days, slight increase; five days, do.

Litmus milk (37° twenty-four hours).—No clotting, decolorised, yellowish colour; three days, quite clotted; five days, do.

Potato (37° twenty-four hours).—Very slight, moist-looking growth; five days, do.

Anærobic growth (37° twenty-four hours).—Turbid, no gas; three days, turbid, no gas.

= *Bacillus para-typhi abdominalis*.

SPECIMEN 2.

September 19th, 1901. H.D. Urine, nil.

G.F.B., 37°. Twenty-four hours; turbid; white; flocculi; H.D., cocci.

Broth, 37°. Twenty-four hours; turbid; no gas.

Made three agar plates; small white colonies.

C.M.B. Staphylococci.

Staphylococci only found.

SPECIMEN 3.—Urine, no albumin.

September 26th, 1901. H.D. Urine, nil.

G.F.B., 37°. Twenty-four hours; abundant gas; marked turbidity; H.D.; cocci, and many motile bacilli.

Broth, 37°. Twenty-four hours; turbid; white sediment; no gas; H.D.; cocci and motile bacilli.

Agar plate. Twenty-four hours; many white colonies; C.F.; staphylococci.

NOTE.—Many cocci and few bacilli in broth, but many bacilli and few cocci in glucose formate broth.

September 27th. Planted three agar plates from glucose formate broth. Two organisms appeared to be present, one simulating proteus, and one simulating an organism of the typhoid group.

Cultures were made from each on agar. They were not pure. Cultures were now made from broth 26th September. Twenty-four hours. Turbidity; H.D.; bacilli, some motile.

Three agar plates were made from this. These were replated twice, and the organism simulating the bacillus typhi abdominalis on agar, was eventually obtained in a pure condition.

This organism when worked out was found to be very like proteus in its reactions.

SPECIMEN 3.—*Urine*, 26th September, 1901.

Description.—A bacillus.

Motility.—Slightly motile (agar twenty-four hours).

Staining.—Faintly with C.M.B., well with C.F. Decolorised by Gram-Weigert.

Pleomorphism.—All forms are short bacilli; practically no pleomorphism.

Broth (37° twenty-four hours).—Turbid, no gas; two days, do.; five days, no indol; eight days, no indol.

Peptone water (37° twenty-four hours).—Turbid, slight sediment, no gas; two days, turbid, no gas; nine days, trace indol.

Formate broth (37° two days).—Good gas, turbid, no colour change.

Dextrose broth (37° twenty-four hours).—Fair gas, decolorised, slight acid; two days, no gas, acid.

Lactose broth (37° twenty-four hours).—Slight turbidity; two days, do.; four days, do.

Saccharose broth (37° twenty-four hours).—Nil; two days, turbid.

Glycerine broth (37° twenty-four hours).—Nil; two days, turbid, slight acid, no gas.

Nitrate broth (37° twenty-four hours).—Turbid, good nitrites, with meta-phenylene diamine.

Lead broth (37° twenty-four hours).—Turbid, good H₂S; two days, very marked H₂S; three days, do.

Gelatin stab (20° twenty-four hours).—Very slight growth; two days, no gas, no liquefaction, white growth on surface; three days, liquefaction at surface; nine days, liquefaction on surface only, horizontally.

Gelatin streak (20° twenty-four hours).—Very slight growth, no gas, no liquefaction; two days, no gas, liquefaction; three days, increased liquefaction; nine days, entirely liquefied.

Gelatin shake (20° twenty-four hours).—A few very small colonies in substance, no gas, no liquefaction; two days, do.; three days, liquefaction on surface; nine days, surface liquefied horizontally.

Gelatin plates (20° twenty-four hours).—Nil; four days, no growth.

Agar streak (37° twenty-four hours).—Vigorous, raised, whitish growth; two days, do.; four days, increased.

Agar plates (37° twenty-four hours).—White colonies; four days, raised white, moist-looking colonies.

Blood-serum (37° twenty-four hours).—White raised, moist-looking growth; two days, liquefaction; three days, do.; six days, complete liquefaction.

Litmus milk (37° twenty-four hours).—Slight acid, no clotting; two days, less acid, no clotting; three days, no clotting, commencing decolorisation; nine days, no clotting, decolorisation, complete precipitation of casein.

Potato (37° twenty-four hours).—Vigorous, yellowish brown, raised, spreading, moist-looking growth; two days, do.

Anærobic growth (37° twenty-four hours).—Turbid, good gas; two days, turbid, fair gas; three days, good gas.

Urine (37° twenty-four hours).—Nil; two days, turbid, no alkalinity.

Approaches bac. proteus vulgaris in reactions.

CASE 12.—F., æt. 30 years. Admitted into Mary Ward on September 4th, 1901, under Dr. Hale-White, for general malaria and pyrexia. She was delivered of a child one month ago. One month ago a lodger in her house died of typhoid fever. She got up after her confinement about a fortnight ago, and caught cold soon after this, and then felt languid and weak and unable to get about. She had a cough. She slept well, but her appetite was bad. The bowels were regular. On September 1st she sent for a doctor who diagnosed enteric fever, and who told her that she had had it ten days.

On admission, temperature 101·6, pulse 132. Tongue furred, abdomen protuberant, walls flaccid, spleen palpable, a few spots on abdomen and lower part of chest. Pulse weak. Heart dulness increased laterally, tick tack rhythm at base of heart. A few râles at bases of lungs behind. Urine 1028, reddish, urates, acid, albumin.

September 5th. Temperature normal. Milk diet. No appetite. Condition unchanged.

September 7th. She feels better. A few fresh spots on abdomen. Temperature 101·6° at night.

September 10th. Dr. Hale-White diagnosed enteric fever.

September 13th. The serum does not give the Widal reaction.

September 18th. Patient progressing favourably. No spots on abdomen. Urine, thick with deposit of mucus. Reaction acid, 1020; no sugar or albumin.

September 24th. Still progressing favourably. Temperature rose to 101·8° yesterday, for no apparent reason.

September 26th. Urine, 1012; cloudy, slight deposit of urates; acid; no albumin.

October 2nd. She is going on well. Temperature 97°.

October 12th. She is still going on well. Temperature 98·4°.

A bacteriological examination of the urine was made on four occasions. On the first, bacillus typhi abominalis with staphylococci were found. On the second, B.T.A. in pure culture. On the third, cocci and some large bacilli not B.T.A. On the fourth, B.T.A. with a proteus-like organism.

Widal reactions :—

September 11th. 50 per cent. = ○. 5 per cent. = ○. 5 per cent. = ○.

September 20th. 50 per cent. = ○. 5 per cent. = $\frac{1}{2}+$. 5 per cent. = ○.

SPECIMEN 1.—No albumin.

September 20th, 1901. H.D. Urine; cocci and slightly motile bacilli.

G.F.B., 37°. Twenty-four hours, turbid; no gas; flocculi.

Broth, 37°. Twenty-four hours, turbid; white flocculi. H.D., slightly motile bacilli.

Agar plate. Twenty-four hours. Cloudiness on plate.

C.M.B. Bacilli (agar). C.F. (broth), *Staphylococci*.

September 23rd. Made three agar plates from agar growth. Nothing grown.

September 25th. Planted three agar plates from broth tube. Twenty-four hours; white colonies. C.M.B., short bacilli. H.D., Motile bacilli. Obtained a pure growth by replating.

Planted agar tube from agar plate.

The organism was worked out according to scheme given below.

SPECIMEN 1. 20th September, 1901.—*Urine*. No albumin.

Description.—A rather short bacillus.

Motility.—Fair motility.

Staining.—Well with C.M.B., fairly with C.F., decolorised by Gram-Weigert.

Broth (37° twenty-four hours).—Turbid, no gas; two days, turbid, no gas; five days, no indol.

Peptone water (37° twenty-four hours).—Turbid, no gas; two days, turbid, white sediment, no gas; five days, no indol.

Formate broth (37° twenty-four hours).—No gas, slightly acid; two days, slight turbidity, slightly acid, no gas; five days, do.

Dextrose broth (37° twenty-four hours).—No gas, acid; two days, no gas, acid; five days, do.

Lactose broth (37° twenty-four hours).—No gas, no colour change; two days, no gas, no colour change; five days, slight turbidity only.

Nitrate broth (37° twenty-four hours).—Good nitrites (with metaphenylene-diamine).

Lead broth (37° twenty-four hours).—No H₂S; two days, slight H₂S; three days, good H₂S; five days, good H₂S.

Gelatin stab (20° twenty-four hours).—Slight growth, no gas; two days, increased; five days, no gas, no liquefaction, growth down stab.

Gelatin streak (20° twenty-four hours).—Slight growth; two days, increased; five days, no gas, no liquefaction, increased growth.

Gelatin shake (20° twenty-four hours).—Turbid, no gas, no liquefaction; five days, turbid, no gas, no liquefaction.

Agar streak (37° twenty-four hours).—Fair, yellowish white, raised growth; five days, do., no spreading.

Blood-serum (37° twenty-four hours).—Raised, yellowish, moist-looking growth; two days, do.; five days, do., drier.

Litmus milk (37° twenty-four hours).—Slightly acid, no clotting; three days, do.; five days, do.

Potato (37° twenty-four hours).—Moist-looking brownish growth; two days, do.; five days, do.

Anærobic growth (37° twenty-four hours).—Slightly turbid, no gas; two days, slightly turbid, no gas.

Gruber's reaction.—Experiment performed with a twenty-four hours' broth culture grown from gelatin slope culture, and with a serum that reacted fully to a known bacillus typhi abdominalis:—

50 per cent. = +. 5 per cent. = +. 5 per cent. = +.

= Bacillus typhi abdominalis.

SPECIMEN 2, 6th October, 1901.—*Urine*, 3rd October, 1901. Acid, slight excess phosphates, no albumin.

Description.—Short oval bacillus.

Motility.—Slightly motile (dextrose two days).

Staining.—Fairly with C.M.B., weak with C.F., decolorized by Gram-Weigert.

Broth (37° twenty-four hours).—Turbid, no gas; two days, do.; five days, no indol.

Formate broth (37° twenty-four hours).—Acid, no gas; two days, acid, no gas; three days, less acidity.

Dextrose broth (37° twenty-four hours).—Acid, turbid, no gas; two days, acid, no gas; three days, do.

Lactose broth (37° twenty-four hours).—Turbid, not reddened, no gas; two days, no gas, no colour change; three days, very slightly alkaline.

Glycerine broth (37° twenty-four hours).—Turbid, not reddened, no gas; two days, do.

Nitrate broth (37° twenty-four hours).—Turbid, no gas, good nitrites (with metaphenylene diamine).

Lead broth (37° twenty-four hours).—Good H₂S.

Gelatin stab (20° twenty-four hours).—Hardly perceptible growth, no gas, no liquefaction; three days, no gas, no liquefaction.

Gelatin streak (20° twenty-four hours).—Very slight growth no gas, no liquefaction, spreading opaque growth.

Gelatin shake (20° twenty-four hours).—Very slight turbidity, no gas, no liquefaction; three days, do.

Agar streak (37° twenty-four hours).—Raised, not very vigorous, greyish white growth; three days, increased.

Agar plates (37° twenty-four hours).—White colonies.

Blood-serum (37° twenty-four hours).—Raised white growth; two days, do.; three days, increased; eight days, do.

Litmus milk (37° twenty-four hours).—Very slightly acid, no clotting; three days, acid, no clotting; eight days slightly acid, no clotting.

Potato (37° twenty-four hours).—Slight, moist-looking growth; two days, do.; three days, growth spreading over surface.

Anaerobic growth (37° twenty-four hours).—Slightly turbid, white deposit, no gas; two days, do., no gas; three days, do.

Gruber's reaction.—This experiment was performed with a twenty-four hours' broth culture grown from a gelatin slope culture, and with a serum which reacted fully to a known bacillus typhi abdominalis.

50 per cent. = 4. 5 per cent. = +. 5 per cent. = +.

= *Bacillus typhi abdominalis*.

SPECIMEN 3.—*Urine*, acid, no albumin.

October 7th, 1901. H.D. *Urine*. Large bacilli; motile.

G.F.B., 37°. Twenty-four hours; turbid; no gas; H.D.; cocci in short chains.

Broth, 37°. Twenty-four hours; turbid; no gas.

Agar plate 37°. Twenty-four hours; growth spreading over plate.

October 8th. Made three agar plates from G.F.B.

October 9th. Planted agar tube from agar plate. Twenty-four hours; greyish white growth of small dotted colonies; H.D.; masses of cocci only; C.M.B.; staphylococci.

In this specimen of urine some large well staining bacilli were seen, but they did not grow—they were much too large for typhoid bacilli. Staphylococci only grew.

SPECIMEN 4.—*Urine*, acid, no albumin.

October 11th, 1901. H.D. *Urine*, nil.

G.F.B., 37°. Twenty-four hours; turbid; sediment; no gas; H.D., slightly motile bacilli.

Broth, 37°. Twenty-four hours; turbid; sediment; no gas.

Agar plate 37°. Twenty-four hours; spreading growth on plate; H.D. slightly motile bacilli.

October 12th. Made three agar plates from G.F.B.

Two days. (1) Proteus-like growth not worked out; (2) White colonies; H.D., motile bacilli; C.M.B., bacilli fairly stained.

October 14th. Planted from white colony.

Broth, 37°. Twenty-four hours; very turbid; no gas; H.D., motile bacilli.

Agar, 37°. Twenty-four hours; greyish white growth; H.D., motile bacilli; C.M.B., well-stained bacilli.

G.F.B., 37°. Twenty-four hours; slight turbidity; H.D., motile bacilli.

The organism having been obtained pure, was worked out from agar culture.

SPECIMEN 4. 15th October, 1901.—Urine, 11th October, 1901. Acid.
No albumin.

Description.—A short bacillus.

Motility.—Fair motility (agar twenty-four hours).

Staining.—Well with C.M.B., faintly and irregularly with C.F., decolorised by Gram-Weigert.

Broth (37° twenty-four hours).—Turbid, no gas; two days, do.; three days, do.; five days, no indol; six days, no indol.

Peptone water (37° twenty-four hours).—Turbid, no gas; two days, do.; five days, no indol.

Formate broth (37° twenty-four hours).—Slightly acid, no gas; two days, do.

Dextrose broth (37° twenty-four hours).—Marked acidity, no gas; two days, do.

Lactose broth (37° twenty-four hours).—No gas; two days, do., slightly turbid, no colour change; four days, no gas, no colour change.

Saccharose broth (37° twenty-four hours).—Slight gas, turbid; two days, turbid; three days, turbid.

Glycerine broth (37° twenty-four hours).—No gas; two days, do., turbid, no colour change; three days, do.

Nitrate broth (37° twenty-four hours).—Good nitrites (with metaphenylene-diamine).

Lead broth (37° twenty-four hours).—Turbid, very slight H₂S; two days, good H₂S; three days, very good H₂S.

Gelatin stab (20° twenty-four hours).—Very slight growth along stab, no gas, no liquefaction; three days, do.; four days, increased growth, no gas, no liquefaction.

Gelatin streak (20° twenty-four hours).—Very slight growth, no gas, no liquefaction; three days, do.; four days, spreading growth, no gas, no liquefaction.

Gelatin shake (20° twenty-four hours).—Turbidity, no gas, no liquefaction; two days, do.; four days, do.

Agar streak (37° twenty-four hours).—Semi-translucent, whitish, raised growth, regular edges; two days, vigorous growth.

Blood-serum (37° twenty-four hours).—Yellowish, raised, moist-looking growth; two days, do.

Litmus milk (37° twenty-four hours).—Acid, no clotting; two days, do.; five days, do.

Potato (37° twenty-four hours).—Moist, yellowish, brown growth; two days, do.; three days, do.

Anaerobic growth (37° twenty-four hours).—No gas formation; two days, do.; three days, do.

Gruber's reaction.—This experiment was done with a twenty-four hours' broth culture growth from a gelatin slope culture, and with a serum which reacted fully to a known bacillus typhi abdominalis.

50 per cent. = 0. 5 per cent. = +. 5 per cent. = $\frac{1}{2}$ +.

= Bacillus typhi abdominalis.

CASE 13.—M., æt, 19 years. Admitted into Stephen Ward on August 19th, under the care of Dr. Fawcett, for pain in the back of the head.

About three weeks ago he began to feel overtired, and had a bad headache on rising; at night time he experienced pain all down his spine. For the past week he has been sweating profusely, and the pain has been much worse, with pain also over the abdomen. He has never at any time lost consciousness, but has been drowsy and apathetic. He has not vomited. The bowels have acted normally. He has had no trouble with micturition. He has seen no spots on his body. He kept at work during the first week of his illness, but then had to give up. He has since gone back to work but could not continue.

On admission, temperature 104.2°, pulse, 116, respiration 24.

He lies in bed with his eyes half closed, and with a very drowsy appearance. He very frequently mutters. The lips are cracked, the gums congested, the teeth foul, tongue thickly furred, breath very foul. The abdomen is full but not distended, is rigid but resonant all over. The liver and spleen cannot be felt. The whole abdomen is very tender to pressure.

The pulse is soft, full, regular, dicrotic, the artery easily compressible. The heart is normal. Respiratory system normal. Urine, 1025; urea, 2.5 per cent., acid, normal.

August 20th. He is wandering in his mind to-day and is very drowsy. He does not complain of abdominal pain to-day. Chloralamide gr. xxx. in brandy.

August 21st. He is drowsy.

August 22nd. He is drowsy, he cannot give a connected answer. Mist. Chloral et Pot. Brom. Urine 1028, acid, high colour, normal.

August 23rd. He answers questions rationally. Splenic dulness enlarged. Abdomen still rigid, but not tender. Tr. Opii. m x. statim.

The serum did not react in any dilution to the Widal reaction.

August 24th. He has had a better night; three typhoid spots on left side of abdomen, two or three on chest.

August 26th. Temperature lower; clot of blood passed.

August 29th. Another specimen taken for Widal reaction.

August 30th. Urine, 1030; acid, urea 3 per cent.; no albumin. The temperature has varied between 100-104° during last four days. Delirium slightly worse. Abdomen less distended.

September 3rd. He is very restless and delirious, temperature 98°-102.4°.

September 10th. He is better this morning; tongue very furred.

September 11th. Urine 1024 alkaline; mucus; no sugar or albumin; mentally weaker; temperature 103.4°.

September 12th. Abdomen rigid though resonant; pain in back and lower limbs.

September 14th. Mental condition slightly improved.

September 16th. Very noisy during night; cough bad; wandering in mind.

September 17th. Still very noisy; sweats profusely; rhonchi in chest; heart rapid; abdomen still rigid. Brandy and I.M.H.

September 18th. He commenced vomiting: keeps nothing down; champagne by mouth; food by rectum; still noisy. Patient died at 6.20 p.m.

Post-mortem examination.—Made 20 hours after death.

Heart. Some recent vegetations on the valves. Otherwise normal.

Lungs. Extensive broncho-pneumonic changes, especially at the right base.

Liver and kidneys normal. Spleen enlarged, some recent capsulitis.

Intestines. Perforation in ileum, six feet from ileo-cæcal valve. Severe ulceration of small intestine, especially near ileo-cæcal valve. Ulcer in cæcum.

The urine of this case was not worked out during life. After death a bacteriological examination was made of the heart-blood, kidney and the urine from bladder. In all of these the bacillus para-colon communis was found in pure culture.

This bacillus differed in its bacteriological reactions from the bacillus coli communis only in the fact that it caused no clotting of milk.

Widal Reaction.

1. August 20th, 1901.

50 per cent. = O. 5 per cent. = O. '5 per cent. = O.

2. August 30th, 1901.

50 per cent. = O. 5 per cent. = +. '5 per cent. = O.

Heart-blood.

Description.—Short thick bacilli.

Motility.—Slightly motile (agar).

Staining.—With C.M.B. and C.F.; decolorised by Gram-Weigert.

Broth (37° twenty-four hours).—Turbid, good gas; two days, turbid, very slight gas; six days, good indol.

Peptone water (37° twenty-four hours).—Turbid, no gas; two days, turbid, no gas; six days, good indol.

Formate broth (37° twenty-four hours).—Turbid, very good gas; two days, turbid, very slight gas.

Dextrose broth (37° twenty-four hours).—Turbid, good gas; two days, turbid, acid, no gas.

Lactose broth (37° twenty-four hours).—Turbid, good gas; two days, acid, no gas.

Saccharose broth (37° twenty-four hours).—Turbid, gas; two days, good gas.

Glycerine broth (37° twenty-four hours).—Turbid, no gas; two days, no gas.

Nitrate broth (37° twenty-four hours).—Turbid, good gas, good nitrites (with metaphenylene diamine).

Gelatin stab (20° twenty-four hours).—Fair growth along stab, no gas, no liquefaction.

Gelatin streak (20° twenty-four hours).—Fair growth.

Gelatin shake (20° twenty-four hours).—Turbid, good gas bubbles, throughout.

Agar streak (37° twenty-four hours).—Vigorous, yellowish white growth, regular edges; two days, increased.

Agar plates (37° twenty-four hours).—Large white colonies.

Litmus milk (37° twenty-four hours).—Acid, no clotting; two days, no clotting; eight days, no clotting.

Potato (37° twenty-four hours).—Moist brownish growth; two days, increased; eight days, increased.

Anaerobic growth (37° twenty-four hours).—Turbid, very abundant gas; two days, very good gas.

= *Bacillus para-coli communis*.

This organism differs from the *bacillus coli communis* in that it does not clot milk and gives no gas with glycerine.

Gruber's reaction.—This reaction was carried out with twenty-four hours' old broth culture grown from gelatin. The serum used gave the following result when used with B.T.A., for Widal's reaction:—

5 per cent. = + +. 0.5 per cent. = + +.

On carrying out Gruber's reaction with the above organism a completely negative result was given, thus:—

50 per cent. = 0. 5 per cent. = 0. 0.5 per cent. = 0.

Urine from p.-m. bladder.

Description.—Short oval bacilli.

Motility.—Slightly, but definitely motile (agar).

Staining.—Stains with O.M.B., well with C.F., decolourised by Gram-Weigert.

Pleomorphism.—Some short forms; short, thick, oval bacilli; longer thick bacilli (C.F.).

Broth (37° twenty-four hours).—Turbid, no gas; three days, do.; seven days, very good indol.

Peptone water (37° twenty-four hours).—Turbid, no gas; three days, do.; seven days good indol.

Formate broth (37° twenty-four hours).—Good gas, turbid, no colour change; three days, very slight gas.

Dextrose broth (37° twenty-four hours).—Good gas; three days, no gas.

Lactose broth (37° twenty-four hours).—Turbid, acid, no gas; three days, acid, no gas, turbid.

Saccharose broth (37° twenty-four hours).—Turbid, no gas; three days, do.

Glycerine broth (37° twenty-four hours).—Turbid, no gas; three days, turbid, good gas.

Nitrate broth (37° twenty-four hours).—Turbid, good gas, good nitrites (with metaphenylene diamine).

Gelatin stab (20° twenty-four hours).—Good growth along stab, none on surface, no gas, no liquefaction; five days, do.

Gelatin streak (20° twenty-four hours).—Good growth, gas on surface, no liquefaction; five days, do.

Gelatin shake (20° twenty-four hours).—Scum on surface, good gas bubbles in medium quarter of an inch below surface to bottom of tube; five days, do.

Agar streak (37° twenty-four hours).—Vigorous, raised, yellowish white growth no gas; three days, increased.

Agar plates (37° twenty-four hours).—Two days, large, round, white colonies.

Litmus milk (37° twenty-four hours).—Acid, no clotting; three days, no clotting; thirteen days, no clotting.

Potato (37° twenty-four hours).—Slight, brownish, moist-looking growth; three days, increased.

Anærobic growth (37° twenty-four hours).—Turbid, very good gas; three days, turbid, slight gas.

= *Bacillus para-coli communis*.

This differs from *bacillus coli communis* only in the fact that it does not cause clotting of milk, and gives no gas with lactose broth.

Gruber's reaction.—This reaction was carried out with a twenty-four hours' old broth culture grown from gelatin. The serum used, gave the following results when used with B.T.A. for Widal's reaction :—

5 per cent. = + +. 0·5 per cent. = + +.

On carrying out Gruber's reaction with the above organism, a completely negative reaction was obtained. Thus—

50 per cent. = 0. 5 per cent. = 0. 0·5 per cent. = 0.

Kidney.

Description.—Short, thick, oval bacilli.

Motility.—Slightly motile, agar two days.

Staining.—Slightly with C.M.B., well with C.F., decolorised by Gram-Weigert.

Pleomorphism.—Short, thick, oval bacilli, some longer forms, some almost like cocci (C.F.)

Broth (37° twenty-four hours).—Turbid, no gas; three days, do.; seven days, good indol.

Peptone water (37° twenty-four hours).—Turbid, no gas; three days, do.; seven days, very good indol.

Formate broth (37° twenty-four hours).—Fair gas, no colour change; three days, no gas.

Dextrose broth (37° twenty-four hours).—Good gas, acid, turbid; three days, do.

Lactose broth (37° twenty-four hours).—Acid, turbid, no gas; three days, do.

Saccharose broth (37° twenty-four hours).—Turbid, no gas, purple colour; three days, do.

Glycerine broth (37° twenty-four hours).—Turbid, no gas; three days, acid, good gas.

Nitrate broth (37° twenty-four hours).—Turbid, good gas, good nitrites (with metaphenylene diamine).

Gelatin stab (20° twenty-four hours).—Good growth down stab, very slight on surface, no liquefaction.

Gelatin streak (20° twenty-four hours).—Whitish growth, no gas, no liquefaction.

Gelatin shake (20° twenty-four hours).—Turbid, gas, no liquefaction, gas not near surface.

Agar streak (37° twenty-four hours).—Well marked, vigorous, yellowish white growth, regular edge; three days, increased.

Agar plates (37° twenty-four hours).—Round, white, large raised colonies.

Litmus milk (37° twenty-four hours).—Acid, no clotting; three days, do nine days, no clotting, acid.

Potato (37° twenty-four hours).—Moist, raised, brownish yellow growth; three days, increased; nine days, increased.

Anaerobic growth (37° twenty-four hours).—Very good gas formation.

= *Bacillus para-coli communis*.

This bacillus differs only from *Bacillus coli communis* in that it does not cause clotting of milk, nor the formation of gas with lactose and saccharose broth.

Gruber's reaction.—This reaction was carried out with a twenty-four hours' old broth culture grown from gelatin. The serum used gave the following result when used with B.T.A. for Widal's reaction:—

5 per cent. = + +. 0.5 per cent. = + +.

On carrying out Gruber's reaction, with the above organism, a completely negative result was obtained:—

50 per cent. = 0. 5 per cent. = 0. 0.5 per cent. = 0.

CASE 14.—M., æt. 24 years. Admitted into Stephen Ward under the care of Dr. Perry, on September 14th, for slight pain in the abdomen and general malaise.

He has been a heavy drinker. His occupation consists mostly of working in drains, and at the time of his illness he was engaged in repairing drains condemned by the sanitary authorities.

Fourteen years ago he had an attack of rheumatic fever, which was followed by another attack five years later.

On August 25th he felt great pain and stiffness in all his limbs, and on leaving work he went to bed. On the next day he saw a doctor, who gave him medicine. On August 27th he went to work, but on August 28th feeling worse he remained in bed. On August 29th he again went to work, and remained at work until September 11th, when he saw his doctor, who told him he had enteric fever, and he was sent into Guy's Hospital. He has suffered a deal from headache and slight giddiness. Until September 11th he had been taking solid food. The bowels have been irregular, at one time being loose, at another constipated, the motions yellowish in colour.

On admission, temperature 102°, pulse, 92, respiration 20. The abdomen moves well on respiration, no spots are to be seen. The spleen is just palpable. Circulatory system: There is a systolic bruit at the apex. Second sound very distinct in the aortic area. Urine, 1025, acid, nothing abnormal.

September 17th. A rose-red spot appeared to-day over right hypochondriac region.

September 18th. He vomited to-day.

September 19th. Four more spots appeared on the abdomen. Another severe attack of vomiting last night.

September 20th. Dr. Perry thought that the systolic bruit was due to an old heart lesion.

September 22nd. He had severe hæmorrhage from the bowel.

September 23rd. Patient had more hæmorrhage and he died at 2 a.m. No Widal's examination was made.

Post-mortem examination.—Lungs congested. Old pleuritic adhesions.

Heart. Old thickening of aortic valves.

Kidneys normal. Liver normal. Spleen enlarged and firm.

Intestines. Ulceration of ileum for about two feet above ilco-cæcal valve. No ulceration of colon. No perforation.

Bacteriological examination of the urine:—

Glucose formate broth, 37°. Twenty-four hours, nil; two days, nil.

Broth, 37°. Twenty-four hours, nil; two days, nil.

Agar plate, 37°. Twenty-four hours, nil; two days, nil.

The urine is sterile.

Bacteriological examination of kidney and spleen.—No satisfactory examination could be made of the kidney, for in spite of all methods of cultivation being employed, no growth could be obtained with enough vitality to bear transplanting. They all died out, both in the kidney and the spleen.

Histological examination of the kidney, stained with carbol thionin blue, shewed no organisms to be present when examined with both one-sixth inch and one-twelfth inch oil immersion lenses.

CASE 15.—M., æt. 16 years. Admitted into Stephen Ward, under the care of Dr. Bryant, on September 9th, 1901, for cough and pain in the abdomen.

He has had cough, together with pain in the abdomen and back; he has been listless and drowsy, and has had shivering fits. On the day before admission he became feverish and lost his appetite.

On admission, temperature, 100·4°; pulse, 100; respiration, 24. He appeared drowsy and inclined to sleep. He complained of pain in the abdomen.

There is a localised apical systolic bruit, otherwise the heart is normal. The spleen is palpable. The abdomen is slightly distended, and there are two or three suspicious spots present. The lungs are normal. Urine 1010, acid; albumin present in small quantity. Diazo reaction obtained.

September 20th. The blood gave a partial Widal reaction.

September 24th. Blood does not react completely to 5 per cent., not at all to ·5 per cent. in the Widal reaction.

September 26th. He developed a sore throat last night, the fauces and uvula being red and inflamed, but no membrane being present. A cultivation was taken, but no Klebs-Löffler bacilli were found. He has fits of coughing and there are râles and ronchi in chest.

September 28th. The blood gives the Widal reaction in 5 per cent., but not in ·5 per cent.

October 1st. Temperature, 100·8°. Urine normal.

October 6th. Temperature, 98·6° in morning, 102° in evening. He feels comfortable.

October 8th. Temperature, 97·8° to 101·6°.

October 10th. Spleen felt below costal margin on inspection. It feels rather hard. The throat has cleared up. Temperature 103·8°.

October 14th. He feels better this morning. Temperature 101·8°. Spleen still palpable below costal margin.

Widal reaction, 5 per cent. = + +. ·5 per cent. = + +.

October 15th. Urine, 1008; no sugar or albumin; acid, urea, 1·2 per cent. Evening temperature 104°.

A bacteriological examination of the blood was made on one occasion. It was found to be sterile.

A bacteriological examination of the urine was made on three occasions, and on each of these it was found to contain only staphylococci.

Widal examinations.

1. 11th September, 1901—
50 per cent. = O. 5 per cent. = O. ·5 per cent. = $\frac{1}{2}+$.
2. 17th September, 1901—
50 per cent. = +. 5 per cent. = O. ·5 per cent. = O.
3. 24th September, 1901—
50 per cent. = ppt. 5 per cent. = $\frac{1}{2}+$. ·5 per cent. = O.
4. 26th September, 1901—
50 per cent. = +. 5 per cent. = +. ·5 per cent. = O.
5. 17th October, 1901—
50 per cent. = . 5 per cent. = ++. ·5 per cent. = ++.

SPECIMEN 1. *Urine*.—No albumin.

September 26th, 1901. H.D. Urine, nil.

G.F.B., 37°. Twenty-four hours, turbid; no gas. H.D., cocci only, chiefly in pairs and groups. C.M.B., cocci in pairs and groups.

Broth, 37°. Twenty-four hours, turbid; no gas.

Agar plate, 37°. Twenty-four hours. A few white colonies.

C.M.B. Staphylococci only.

SPECIMEN 2. *Urine*.—Acid, slight albumin.

September 28th 1901. Broth, 37°. Three days, turbid.

Agar plate, 37°. Three days, nil.

October 1st. Planted broth, 37°. Twenty-four hours, turbid; no gas. H.D., cocci only, chiefly in pairs.

October 3rd. Planted three agar plates from G.F.B. Twenty-four hours; dotted white colonies. H.D., cocci only.

C.M.B., Staphylococci only.

SPECIMEN 3.—*Urine*. Acid, slight trace albumin.

G.F.B., 37° twenty-four hours. Slightly turbid, no gas. H.D. cocci only, in pairs.

Broth, 37° twenty-four hours. Turbid, no gas. H.D. cocci, chiefly in pairs.

Agar, 37° twenty-four hours. Small white colonies. H.D. cocci only.

C.M.B., staphylococci only.

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A CARDIOGRAPHIC TRACING, SHOWING ASYNCHRONOUS ACTION OF THE VENTRICLES.

BY THEODORE FISHER, M.D.
PATHOLOGIST TO THE BRISTOL ROYAL INFIRMARY.

THE accompanying cardiographic tracing has been lying amongst my notes for some years. It has always seemed to me to possess features of considerable interest, but since the tracing is accompanied by very incomplete notes of the case from which it was taken, I have not deemed it worthy of being brought before the notice of others. Yet, meagre though the facts connected with it may be, the rarity of such a tracing I trust will be thought sufficient to justify its publication.

When I was taking the work of Dr. Michell Clarke at the Bristol General Hospital, during his summer holiday in 1895, a woman, aged 25, was admitted suffering from cardiac disease. There was œdema of the legs, and the liver was several inches below the ribs. The heart was enlarged, and the apex was apparently in the fifth intercostal space, one inch external to the nipple line, but the special features of interest were in the character of the impulse. My notes thus describe it:—“*The impulse is well marked, and is visible and palpable over the greater part of the cardiac area. At the apex there is a short presystolic thrill, but the most noteworthy feature is the double character of the impulse. There is a distinct double shock, and a striking point is the fact that the second impulse is very well-marked between the*

apex and the sternum. The rise at the apex is first seen, then a rise nearer the sternum. In both places a double shock is felt by the hand, but the earlier shock is most marked at the apex, and the second nearer the sternum."¹ Tracings were taken by means of Marey's cardiograph, which clearly illustrated the movements felt and seen. The button of the tambour of one lever was placed at the apex and the button of the tambour of the other was placed at a spot one inch from the left border of the sternum, also in the fifth interspace. Simultaneous tracings of the movements at these two points were thus obtained, and a reproduction of these tracings is shewn in the accompanying illustration. The upper tracing is from the apex, the lower from the point near the sternum. It will be noticed that the higher waves in the tracing from the apex are not situated immediately over the high waves of the tracing from the point near the sternum. There are other details of interest, but this is the most striking characteristic of the tracing.

We may reasonably, I think, attribute the undulations of the upper tracing to movements of the left ventricle, and at least the higher waves of the lower tracing to movements of the right ventricle. I am aware that the power of the right ventricle to give rise to a tracing has been doubted, and it is possibly true that most tracings obtained from the surface of the right ventricle merely indicate transmitted movements of the left ventricle. In mitral stenosis, of which disease this case was an example, there is, however, often considerable hypertrophy of the right side of the heart. But although there was not an opportunity of discovering that an amount of hypertrophy of the right ventricle existed in this case which could easily have given rise to a tracing, it cannot be supposed that the left ventricle was capable of producing at one time a high wave outside the nipple line and at another a similar wave close to the sternum. If the tracing from the apex indicated movements of the left ventricle, then the other tracing must have recorded movements of some other

¹ It may be of interest to add that the patient was conscious of a peculiar cardiac action, and stated that it had commenced a few days before admission, during a severe thunderstorm.

contractile body, which in this instance could have been nothing else but the right ventricle. This seems to be indisputable so far as the higher elevations are concerned, but it is possible that the minor curves record impulses transmitted from the left ventricle. If this be considered to be the most reasonable explanation of the minor curves, the tracing becomes still more interesting, because we shall then have to believe that the right ventricle intermitted while the left was contracting. Putting on one side, however, the question of the mode of production of these minor curves, examination of the tracing from the apex shows that a comparatively forcible systole of the left ventricle alternated with one much more feeble, and on comparing the tracing with that taken near the sternum it will be noticed that a strong contraction of the underlying ventricle generally took place about the same time as the weaker contraction of the left ventricle. At one spot the left ventricle apparently intermitted when the right ventricle was contracting, and towards the end of the tracing four strong systoles of the right ventricle have occurred to two of the left.

Unfortunately I have lost my record of the cardiac sounds. I must have examined the case on several occasions, since I have several pulse-tracings taken at different times, both from the radial and the carotid arteries, and also other tracings from the heart. Not only have my notes been mislaid, but I regret to say that I have no recollection of what was heard. Brief notes occur on one of the cardiographic tracings, but they occur at a time when the curious impulse had disappeared, and have evidently been made there as a memorandum of the fact that the cardiac sounds had altered. The date of the disappearance of the abnormal impulse was six days after the first tracing had been taken. The apex-beat then presented nothing especially noteworthy, and the sounds heard there, were a "systolic murmur, loud second sound, with a rumbling and blowing diastolic murmur." At the point close to the sternum where the second tracing was taken the sounds were a "systolic murmur, loud second sound, and a diastolic sound." That is to say, what some would describe as a false reduplication of the second sound was associated with the systolic murmur. A

subsequent note also shows that the general features were merely those of the failing heart of mitral stenosis. The double action is mentioned as absent and "a thrill and short shock" as being felt. The sounds are also described as resembling—

{ therr	{ dup	{ durr
{ Systolic murmur.	{ Second sound.	{ Diastolic murmur,

occasionally varied with a "rapid, rolling action," represented by "therrop, therrop, therrop." It should be mentioned that when the double cardiac action was present, only one of the two beats was felt at the wrist. The pulse was then slow, from forty-four to fifty-two to the minute, but when the curious cardiac rhythm disappeared it rose to ninety-two to the minute. The accompanying tracings, taken from the right carotid artery—one at the time the anomalous cardiac action was present, and the other after it had passed off—give some idea of the alterations in the pulse-rate. The first tracing also shows that only the stronger of the two contractions of the left ventricle made itself felt in the arteries.

Although I have no record of the sounds of the heart when the double impulse was present, it may be safely asserted that they did not appear to me to throw light upon the question of the causation of reduplication of the first sound. At that time I was interested in reduplication of the first sound, and in the bruit de galop, and shortly after wrote a paper on the bruit de galop (*American Journal Med. Sciences*, Sept., 1896). Had the cardiac sounds in this case manifested features bearing upon the subject, I should have remembered what was present. It is possible, however, that the fact that the first sound was replaced by a systolic murmur, and that a presystolic murmur was associated with it, may have made it difficult to appreciate lessons which might otherwise have been taught.

In cases of well-marked reduplication of the first sound and of the bruit de galop, a double shock is easily felt by the hand placed over the impulse. This doubling of the impulse can be recorded by means of the cardiograph, but it is not always easy to obtain a tracing, partly because the impulse is generally feeble and diffuse, and partly because very slight pressure depresses or obliterates the first of the two waves. I have sometimes found

Dudgeon's sphygmograph give a better idea of the movements felt by the hand than Marey's cardiograph.

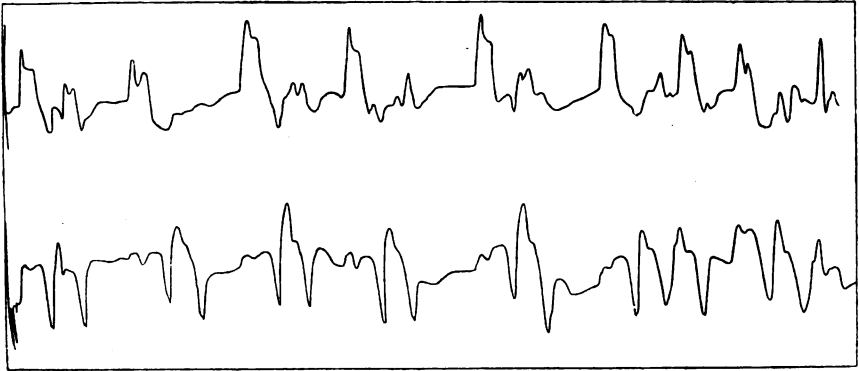
A few tracings are given to illustrate the features of the impulse associated with a bruit de galop and a reduplicated first sound. Other tracings accompany those taken from the same cases at times when the bruit de galop or reduplicated first sounds were absent. In the tracing from the case of a bruit de galop, a high wave is seen to precede the ventricular systolic wave, and a broader wave occupies the same position in the tracing from the case of a reduplicated first sound. In the case of reduplication of the first sound, there are tracings taken at two positions, one at the apex, the other in the fourth space one inch nearer to the sternum. It is interesting to note that in the tracing taken internal to the apex, the situation of the two waves is reversed. At the apex the second wave is the higher, but towards the sternum the first wave rises above the second. What this indicates it is difficult even to suggest. It has not been intended to discuss the causation of reduplication of the first sound. These tracings have been brought forward merely to show that a doubled impulse is present in cases in which a bruit de galop, or a reduplicated first sound is heard. It may be stated, however, that it has in the past not seemed to me necessary to think that the general condemnation of the view, which attributes reduplication of the first sound to asynchronism of the ventricles, has been a faulty judgment. In my paper upon the bruit de galop, which bruit it is scarcely necessary to remark is very closely related to reduplication of the first sound, I have attributed the high wave that precedes the ventricular systolic wave to systole of the auricles. The subject, however, presents many difficulties, and it is one upon which one cannot dogmatise. The abnormal rise in the tracing from a case of reduplication of the first sound, cannot I think be attributed to systole of the auricles. A possible explanation of the tracing would be that asynchronism was present in the contraction of the ventricles, and that the higher part of the doubled wave was produced by the left ventricle at the apex, but by the right ventricle nearer the sternum. It may be remarked that if this explanation be allowed, the case

lends support to Dr. Gibbes's (*Lancet*, vol. i., 1901, page 1601), theory of the causation of the presystolic murmur, which theory attributes the murmur to partial asynchronous action of the ventricles. When the patient, a girl aged twenty, was first seen, a rumbling presystolic murmur, associated with a thrill, was audible at the apex. Three days later, when an abnormal impulse which felt "like two waves passing under the hand in quick succession," was found to be associated with a reduplicated first sound, the presystolic murmur had disappeared. Adopting the theory of Dr. Gibbes, we may suppose that at first there was sufficient asynchronism to give rise to a thrill and a presystolic murmur, and that later, when the asynchronism increased, the thrill became analysed into a double impulse, and the presystolic murmur differentiated into one sound and part of another. Unfortunately for this theory, however, the tracings taken before the reduplication of the first sound shewed itself, and also after it had disappeared, give little indication of asynchronism of the contraction of the ventricles; yet possibly, one tracing in places may be thought to slightly suggest it.

Whatever may be one's view of the causation of a reduplicated first sound it seems to me that writers upon the subject go too far when they strongly assert that asynchronism of the ventricular contractions is not possible. Mackenzie, in his able and comprehensive paper upon the liver and venous pulses (*Journal of Pathology*, vol. ii., 1894, pages 112 and 318), gives tracings which seem to prove conclusively that such asynchronous action can occur. He has taken tracings simultaneously from the internal jugular vein and from the carotid artery in a large number of cases, and one tracing shews pulsations in the jugular vein of ventricular type continuing while the carotid pulse intermits, and another reveals remarkable discordance of the two sides of the heart.

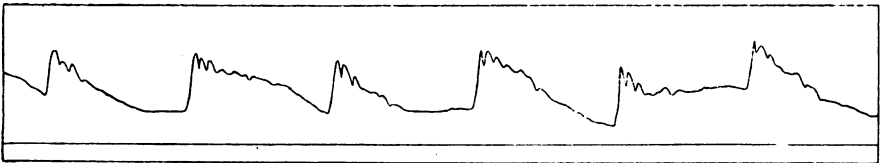
The first tracings which accompany this paper seem to me also to prove conclusively that asynchronism may be present at least in the force of the contractions of the two ventricles.

Fig. 1.



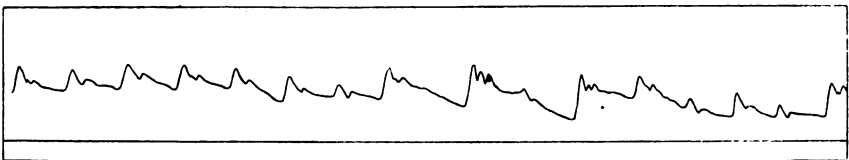
Tracings taken with Marey's cardiograph simultaneously at the apex in the 5th intercostal space one inch outside the nipple line, and at a point one inch from the left border of the sternum, also in the 5th intercostal space. The high curves on the lower tracing do not underlie the higher undulations on the upper tracing.

Fig. 2.



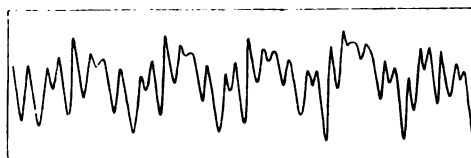
Pulse tracing from the right carotid artery when the double impulse was present. It gives some idea of the slowness of the pulse, and shows that the alternating character of the cardiac contraction was not communicated to the arteries.

Fig. 3.



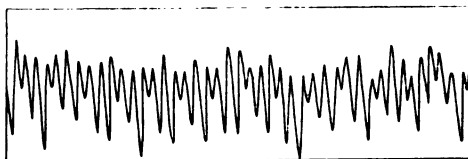
Pulse tracing taken from the same artery seven days later, when the unusual features of the impulse had disappeared. The pulse, when this tracing was taken, had nearly doubled in frequency.

Fig. 4.



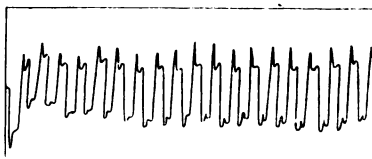
Tracing of heart impulse taken with Dudgeon's sphygmograph when a bruit de galop was present. Respiratory undulations are also present. A high pointed curve precedes the broader undulation due to systole of the ventricles.

Fig. 5.



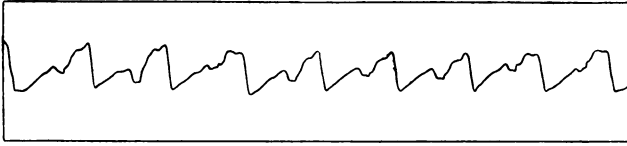
Tracing from the same heart ; but the breath held, and the paper running more slowly.

Fig. 6.



Tracing taken five months previously with Marey's cardiograph when no bruit de galop was present.

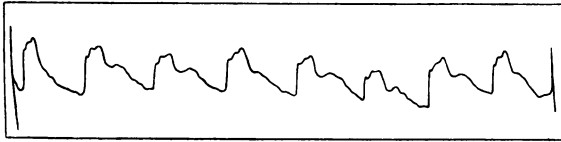
Fig. 7.



Tracing taken at the apex in the 5th interspace, $3\frac{1}{2}$ inches from the left border of the sternum, in a case of reduplication of the first sound.

(Marey's cardiograph.)

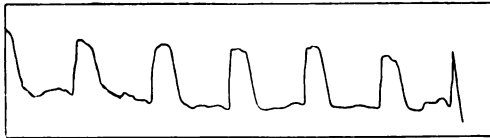
Fig. 8.



Tracing from the same case taken in the 4th inter-space one inch nearer the sternum.

(Marey's cardiograph.)

Fig. 9.



Tracing from the same case taken at the apex when the reduplication of the first sound had disappeared.

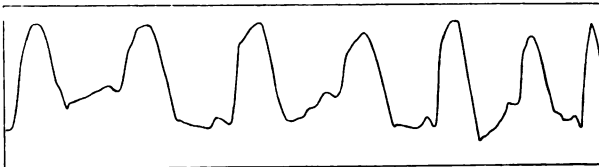
(Marey's cardiograph.)

Fig. 10.



Tracing taken with Dudgeon's sphygmograph when the eduplication of the first sound was present.

Fig. 11.



Tracing taken with Dudgeon's sphygmograph three days previously at the apex when the reduplication was absent, but a presystolic murmur was audible.

ON ACUTE SEPTIC INFLAMMATION OF YOUNG BONE GROWING FROM CARTILAGE.

BY R. LAWFORD KNAGGS, M.C. CANTAB,

ASSISTANT SURGEON TO THE LEEDS GENERAL INFIRMARY.

THERE are few diseases more perplexing to the student than those connected with septic inflammation of bone. The subject is usually split up in such a way that the learner often fails to perceive that any very definite connection exists between conditions which are in reality the various complications or results of a single disease. He may indeed be pardoned if he finds it impossible to unravel the true story from the articles prepared for his consumption under such headings as "Acute Diffuse Periostitis," "Acute Infective Osteomyelitis," "Acute Necrosis," "Acute Epiphysitis," "Acute Arthritis," and "Acute Arthritis of Infants." Nor are his difficulties made easier by the fact that the most recent editions of the leading text-books are not all up to date.

Much of the haze which surrounds the subject is due to a slavish adherence to the lines upon which it was formerly taught. But by degrees well known conditions have been traced back to their source, and now that the cause is found to be the same in all, the time has surely come to study the disease as a whole by following it from its commencement to its terminations.

U. O. N.

This is the object of the present paper. The writer does not claim to say anything new, though he may hope here and there to bring into prominence certain facts which have not as yet received adequate recognition, and above all to simplify what is admitted to be, a very difficult subject.

The title adopted is necessarily comprehensive, but at the same time a limiting one. The conditions dealt with always occur in young people before ossification is complete, and take their origin at some spot where cartilage is being transformed into bone. Consequently septic inflammation of bone in adults is not within its scope, and will only be referred to incidentally or for purposes of contrast.

BACTERIOLOGY.

Though this portion of the subject has not been investigated by the writer, it should be mentioned that the disease is generally believed to owe its origin to pyogenic organisms. Of these the most common is the *staphylococcus pyogenes aureus*, but further information on this point may be found in the text-books of Erichsen (vol. ii., p. 278), and Rose and Carless, and in an experimental paper by Tubby in the *Guy's Hospital Reports* for 1890 (vol. 47, p. 85).

It is probable that in some instances the micro-organisms find an entrance into the blood through some skin lesion, but the respiratory and alimentary tracts are also open to them, so that it is not surprising that the source of the infection is not always obvious.

THE PRIMARY FOCUS.

In the long bones this is situated in one of two places; either—

(1) On the diaphysial side of the epiphysial line, or of the cartilaginous epiphysis; in other words in the juxta-epiphysial region (Figs. 2 and 3); or

(2) At the edge of an ossifying centre in the cartilaginous epiphysis (Fig. 19).

In the small cancellous bones it occurs—

(3) At the periphery of the centre of ossification (Fig. 1).

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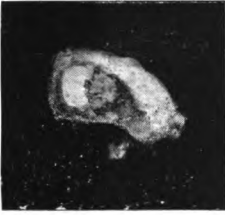


FIG. 1.—From C. L. S., æt. two months, admitted with an acute abscess in the neighbourhood of the ankle-joint, and who died in hospital from convulsions. The photograph shows a cartilaginous astragalus, containing an abscess cavity, in which the necrosed centre of ossification lies loose.

FIG. 1.



FIG. 2.

FIG. 2.—Letitia G., æt. 16. Sub-periosteal abscess of tibia. Pyæmia. Death. A vertical antero-posterior section has been made through the upper part of the tibia. The epiphysis is almost completely united to the diaphysis; but a small portion of the epiphyseal cartilage still persists on either side. Beneath the anterior portion a small abscess cavity is seen, which communicated with the sub-periosteal abscess on one side of the tubercle of the tibia. The insertion of the ligamentum patellæ is seen above it, and is suggestive of the part that structure may have played in the production of the disease.

In one instance it was found beneath the articular cartilage of the acetabulum (Fig. 22), but as the child was only six months old the small abscess cavity at that spot was really at the advancing edge of the enlarging ossific centre.

In every instance in which the writer has had the opportunity of examining bones the subject of this disease, either in consequence of death or after amputation, the starting point of the mischief was discovered, or could with certainty be inferred when the changes were not too far advanced for it to be demonstrated; and in no case was there the least reason to suppose that the periosteum had played any other than a secondary part. It is, however, important that the bone should be examined soon after removal, as a small patch of suppurative osteitis which could not possibly be overlooked when the surrounding cancellous tissue is of the usual red colour, soon becomes difficult to distinguish when the fluids, in which the bone is preserved, have decolourized the specimen.

PREDISPOSING AND EXCITING CAUSES.

These may be divided into two groups. In one may be included such conditions as the patient's general health, or his power to resist infection, the local lesion from which the blood infection takes place, the more general pyæmic state of which, in some cases, the bone lesions may be expressions, and the proneness of the highly vascular growing tissue at the juxta-epiphysial region to become the seat of inflammatory changes. These will not be discussed.

The other comprises certain local anatomical and surgical conditions, which have a very direct influence upon the production of the disease. They are:—

1. The juxta-epiphysial region.
2. The influence of ligament and tendon strain.
3. Traumatic separation of an epiphysis.

1. *The juxta-epiphysial region.*—In the long bones the junction of the diaphysis with the cartilaginous epiphysis, or with the line of cartilage which is interposed for a number of years between the largely ossified epiphysis and the diaphysis, is so well known to be a weak spot in the continuity of the bone that it hardly

requires a reference to the frequency with which separation of the epiphysis is met with in children to emphasize it. This line of weakness, and of cleavage in cases of separation, is not the actual epiphysial line, but the soft, spongy layer of bone uniting the diaphysis with the conjugal cartilage. It has already been alluded to as the juxta-epiphysial region.

Injuries of an indirect nature, such as ricks, twists and sprains in which leverage comes largely into play, but in which the force is insufficient to cause fracture, or separation of the epiphysis, are more likely to lead to traumatism at this spot than at any other part of the bone.

Into the epiphysis are inserted ligaments and strong tendons, which, when put upon the stretch by strain or muscular action, serve to fix it, so that the stress of the injury falls upon the bone between the points of application of the force and counter-force, and the juxta-epiphysial region, in consequence of its weakness, being less able than any other part of the bone to withstand the tearing force brought to bear upon it, is prone to suffer.

2. *The influence of ligament and tendon strain.*—In many of the illustrations accompanying this paper there is much that is extremely suggestive of the part played by ligaments and tendons in the production of the initial lesion.

Thus, Fig. 22 shows a small abscess cavity just below the insertion of the ligamentum teres into the acetabulum. Another photograph (Fig. 5) shows the epiphysial cartilage separated (by suppuration) from the lower end of the diaphysis of the tibia on that aspect which is enclosed by the anterior and posterior inferior tibio-fibular ligaments, and the separation is just below the tibial insertion of the inferior interosseous ligament. In Fig. 2 the cavity of a small abscess which led to acute periostitis is situated just underneath the anterior extremity of an epiphysial line which is completely ossified in its central portion. The ligamentum patellæ, which is also shown, would so obviously pull upon and tend to strain the weak spot, that the influence it probably exerted in the production of the disease can be appreciated at a glance.

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FIG. 3.

FIG. 3.—R. G., æt. 13, six days before admission was kicked behind the right knee, and in trying to save himself he jerked his knee. There was acute subperiosteal abscess of the femur and acute arthritis of the knee-joint. Pyæmia appeared within a few hours of the incisions. The right hand specimen shows a small abscess cavity (bristle) at the juxta-epiphyseal line, and an indistinctness in the cartilaginous disc in its vicinity. In the left hand specimen the way in which the pus found its way beneath the periosteum of the diaphysis on the posterior surface is seen (bristle). Areas of osteitis are present in the diaphysis, the most marked being in direct contact with the abscess. The attachment of the posterior ligament of the joint to the epiphysis is also shown.



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FIG. 4.

FIG. 4.—Alfred P., æt. 16. Admitted with a history of five weeks' illness, and a sub-periosteal abscess, and acute arthritis of the left knee. The limb was eventually amputated. The specimen shows necrosis of a portion of compact bone and of a certain amount of cancellous tissue. The chief disease is on the posterior surface and in close proximity to the epiphyseal line, with which the disease was in direct contact in the other half of the bone. The epiphyseal line cannot be traced in its posterior half in the photograph as it was softened and had lost the appearance of cartilage. The outline of the periosteal abscess is seen, and also some erosion of cartilage on the condyle.

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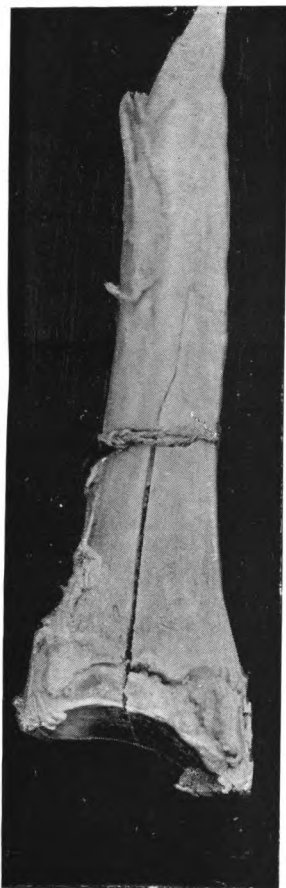


FIG. 5.

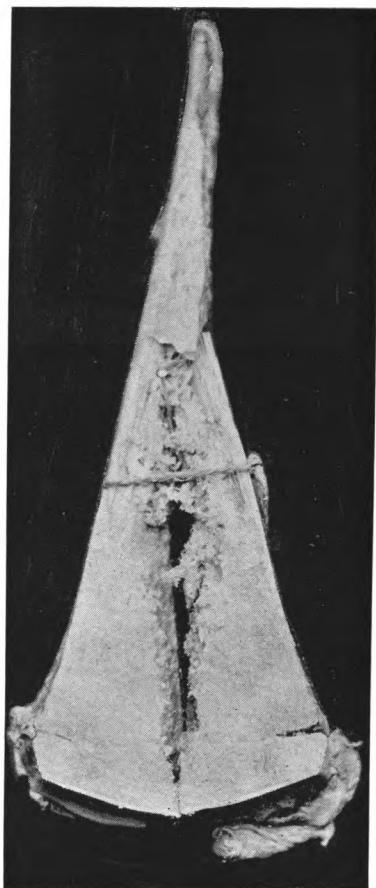


FIG. 6.

FIG. 5 and FIG. 6.—Ambrose H., æt. 10. Death from pyæmia. The photographs show the lower extremity of a tibia. The mischief has begun at the outer part of the juxta-epiphyseal line in the immediate vicinity of the inferior tibio-fibular joint and the inferior interosseous ligament. Pus has tracked upwards, and led to a sub-periosteal abscess of the diaphysis, and downwards beneath the articular cartilage of the inferior tibio-fibular joint, entering the ankle-joint, and causing suppurative arthritis.

(By permission of Dr. CHURTON).

The frequency with which the bones which enter into the knee-joint are attacked in this disease would seem to indicate the existence of conditions favourable to its development. Now, the knee can be fully flexed upon the thigh without putting its ligaments unduly upon the stretch, but extension is checked by all the ligaments except the patellar. Especially may be mentioned the posterior crucial and the posterior ligament of Winslow, and with them may be included the tendon of the semi-membranosus, and the two heads of the gastrocnemius. All these are inserted into the two epiphysis entering into the knee-joint, and when put upon the stretch in over extension the tendency of their pull is to tear apart the epiphysis and the diaphysis of one or other bone at its posterior aspect.

All the information that can be gathered from the specimens here figured, points strongly to the belief that the primary focus about the knee is produced through these agencies, and if so, it explains why it is that the common place for necrosis in the lower end of the femur is behind, in the triangular flat space above the condyles.

3. *Traumatic separation of the epiphysis.*—If injuries to the epiphysial line short of separation are frequent exciting causes of the disease, it might be expected that actual separation would also figure in that capacity. Fig. 7 is taken from a specimen in which traumatic separation of the lower epiphysis of the ulna no doubt determined suppuration, and other examples may be found among the cases recorded in Mr. Poland's work on Traumatic Separation of the Epiphysis.

THE COMPLICATIONS OR CONSEQUENCES.

The initial lesion whose causation and situation have been discussed, is a small suppurating focus of a more or less intensely irritating character. The conditions to which it may give rise depend upon the place where it forms, and upon the direction, or directions, in which the pus may extend. The latter is influenced by the nature of the tissues by which the abscess is bounded, and also by the virulence of the poison.

The conditions which may directly result from acute septic inflammation of bone growing from cartilage are these :—

- | | |
|--------------------------------|---------------------|
| 1. Acute subperiosteal abscess | } of the diaphysis. |
| 2. Acute osteomyelitis ... | |
| 3. Necrosis. | |
| 4. Separation of an epiphysis. | |
| 5. Acute septic arthritis. | |
| 6. Acute deep-seated abscess. | |

I. ACUTE SUBPERIOSTEAL ABSCESS.

In the long bones the deep or fibrous layer of the periosteum is attached to each epiphysial line, and also to the articular cartilages at both ends. It is possible, therefore, for pus to accumulate under the periosteum covering the diaphysis, or under that portion of it which overlies an epiphysis. An acute subperiosteal abscess of the epiphysis does occur, but is probably never recognised clinically, for in addition to its small size it is under cover of tendons and ligamentous expansions, and the pus tracking towards the joint, lifts up the articular cartilage, bursts into the joint and leads to arthritis (Fig. 16).

The subperiosteal abscess (acute infective periostitis) which is so well known, is situated under the periosteum of the diaphysis. When this is uncomplicated the original focus will frequently be found just at that portion of the juxta-epiphysial line which abuts upon the surface of the bone, usually, in the case of the knee, the posterior surface (Fig. 8).

In these cases the juxta-epiphysial strain is probably received during hyperextension, and the situation of the initial lesion is explained by the fact that the maximum injury will be at that part of the line which is first exposed to the tearing strain. The pus, therefore, has not far to travel before it reaches the under surface of the periosteum. Here it finds an easy path of least resistance, and the periosteum, irritated by its virulent character, becomes secondarily inflamed, and no doubt is responsible for a very large proportion of the pus which is evacuated when the abscess is incised.

As the implication of the bone in the vicinity of the conjugal cartilage may under such circumstances be very trifling, these

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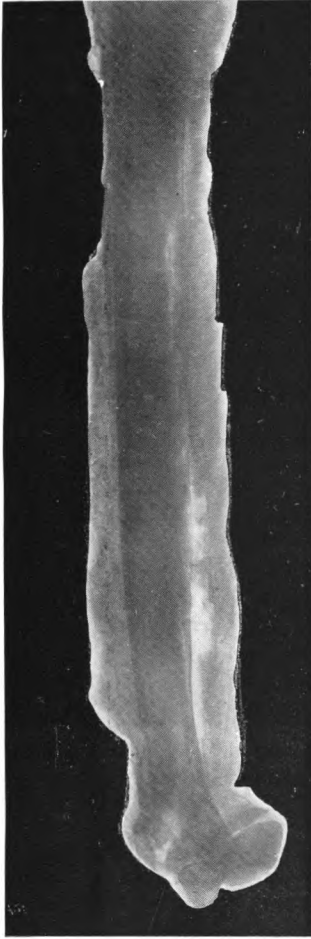


FIG. 7.

FIG. 7.—A boy received an obscure injury to his wrist from a fall. A subperiosteal abscess developed in a few days. The elbow-joint became involved, and eventually amputation was necessary. The specimen shows that the original injury was a separation of the lower epiphysis of the ulna. New periosteal bone is seen reaching down to, and helping to fix the epiphysis. The elbow-joint was implicated by the pus tracking between the insertion of the brachialis anticus and the origin of the flexor carpi ulnaris.

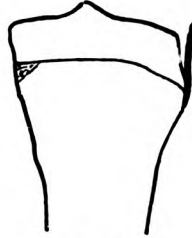


FIG. 8.

FIG. 8.—A diagram to represent what is probably a not uncommon situation of the primary focus at the posterior part of the juxta-epiphyseal region of the head of the tibia. The writer has seen several instances of this lesion; but the changes due to preservative fluids have spoilt the specimens for photographic purposes. Such specimens are, as a rule, only obtained after death from pyæmia, as the bone lesion is too trivial to necessitate amputation.

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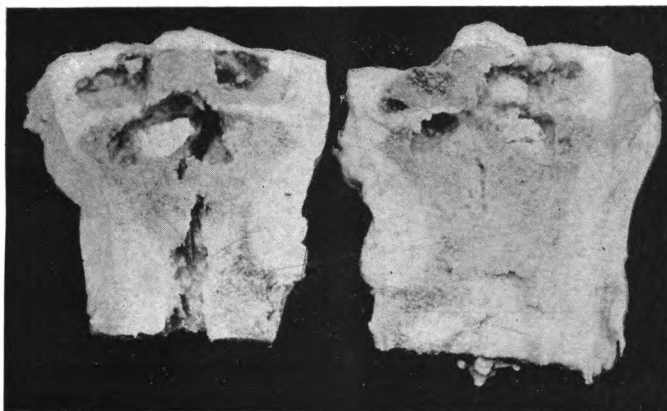


FIG. 9.

FIG. 9.—Upper portion of a tibia sawn open—from a leg amputated for suppurative arthritis and its consequences. The central portion of the epiphyseal disc has been destroyed. From this point the inflammatory process has extended in all directions leading to absorption of cancellous tissue and small areas of necrosis. (The soft granulation tissue has been carefully removed). The epiphysis has been invaded, and the process has extended through the bone until the articular cartilage has been reached and the joint involved. The upper end of the diaphysis has suffered considerably and the medullary cavity has just been reached. In the left hand specimen a glass rod shows a connection between the large central cavity and a cavity in the epiphysis, and another shows the track along which the disease has extended to the medulla. In the right hand specimen the process shows more lateral extension, but no subperiosteal abscess was formed. The infection has evidently not been of the most virulent type for the extension of the disease has been retarded long enough to ensure the removal of a good deal of bony tissue, and the medulla, though reached, was not obviously affected.

(By permission of Mr. W. H. BROWN).

cases may clear up, if an early incision is made, without any obvious indication of necrosis; any small area of bone that may die coming away as granular debris or being absorbed by the resulting granulation tissue.

2. ACUTE OSTEO-MYELITIS.

If this term is applied to an acute inflammation of the peripheral extensions of the medullary tissue that fill up the spaces in the cancellous ends of the diaphysis, or of the soft tissue of the cancellous spaces of the epiphysis, then no doubt acute osteomyelitis is present in every case to some extent, but always extending from the initial lesion. But acute suppurative inflammation of the medullary cavity is one of the least common of the sequelæ of the inflammation of growing bone. Only one of the specimens that the writer has had the opportunity to dissect (Figs. 10 and 11) illustrates this condition; but another specimen (Fig. 9) shows how it is brought about, and why it is not common.

The acute inflammatory process, as it extends from the central point of primary infection, meets with different tissues, which oppose varying degrees of resistance to its progress. At last the suppurative process opens up a space in which the resistance is comparatively trifling, and immediately extension takes place rapidly in this direction, and a relief is afforded by which extension in other directions is brought practically to a standstill.

Thus, as the area of purulent infiltration increases, it may reach either the surface of the bone under the periosteum, and give rise, as already stated, to acute subperiosteal abscess, or it may open up the joint and give rise to acute arthritis, or it may extend to and infect the medullary cavity. Now, unless the infection is exceedingly virulent, the medullary cavity is in many cases the most difficult of the places for the process to extend to, because the developed cancellous bone is less easy of solution than the zone of growing tissue below the conjugal cartilage, or than the cartilage itself, and an opposing barrier is more readily formed. Consequently, either acute subperiosteal abscess or acute arthritis is apt to develop before the medulla is invaded, and

the tension of the inflammatory products within the bone being relieved, extension towards the medulla is checked.

The nearer the centre of the juxta-epiphysial region the primary seat of infection is situated, the greater is likely to be the progress towards the medullary cavity. This is well exemplified in Fig. 9; and another specimen, already alluded to (Figs. 10 and 11), in which the central medulla was infiltrated with pus throughout, confirms it. The bone, for which the writer is indebted to the kindness of his colleague, Mr. Littlewood, shows, on longitudinal section, that the whole of the central part of the upper epiphysial disc has been destroyed, and that there has been extensive necrosis of the cancellous tissue extending towards the medulla, yet the small size of the periosteal abscess and the persistence of the peripheral parts of the cartilaginous disc indicate the late arrival of the pus beneath the periosteum and the freedom of the peripheral parts of the cartilage from inflammatory softening.

But in those cases in which the primary lesion abuts on the surface of the bone, the pus is quickly discharged beneath the periosteum, and extension towards the central parts of the bone can hardly be said to take place at all. Now, as the peripheral part of the juxta-epiphysial line is very commonly affected, and in cases of strain or injury the most likely place to suffer, the infrequency of suppuration of the central medulla is not a matter for astonishment.

Though these points are of considerable interest in connection with the development or prevention of suppuration in the central medulla, there is one other that transcends them all in importance, viz., the degree of virulence of the infection. As in other infections, the more virulent the poison the less able are the vital powers of the patient to offer opposition to its spread. The difference in virulence can best be illustrated by comparing the effects produced in two cases—

Specimen 1218 F in the Royal College of Surgeons' Museum is the lower half of the right femur of a girl of eleven, who was admitted into a hospital with a large abscess at the lower end of the right thigh. The bone was found to be acutely inflamed, and

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FIG. 10.



FIG. 11.

FIG. 10 and FIG. 11.—W. H., æt. 15, a labourer, had a subperiosteal abscess opened a fortnight after a kick at football, which had led first to swelling and then to very severe pain in the leg and constitutional disturbance. Abscesses subsequently formed about the ankle, and amputation was performed.

FIG. 10 is an antero-posterior section of the bone. The central medullary cavity was infiltrated with pus throughout. Below the upper epiphysial disc a very considerable area of the cancellous portion of the diaphysis was necrosed and partially separated. This necrosed portion did not reach to the peripheral extremities of the conjugal cartilage. The destruction of tissue seen in the photograph below this was due partly to a fracture caused in sawing open the bone, and partly to the gouging of the bone when the periosteal abscess was opened. The primary focus was probably near the centre of the juxta-epiphysial area, and infection had spread rapidly towards the central medulla and involved it before pus reached the surface of the bone and led to the subperiosteal abscess, whose small size is well shown in FIG. 11.

(By permission of MR. LITTLEWOOD.)

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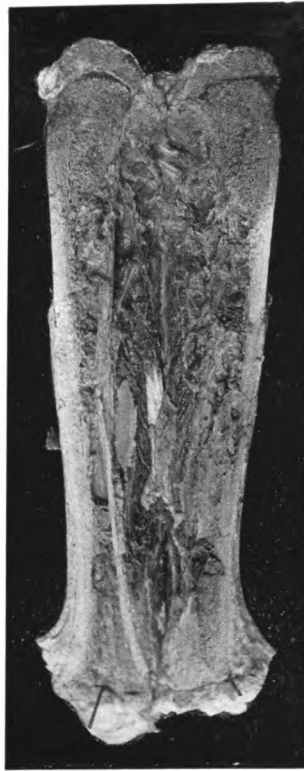


FIG. 12.

FIG. 12.—Acute osteomyelitis of the tibia of a boy about twelve years old. Amputation.

The most pronounced changes are at the upper extremity of the diaphysis, which is completely separated from the epiphysis and necrosed. The medulla was infiltrated with pus, and the juxta-epiphysial region at the lower end of the diaphysis had been reached, and separation of the lower epiphysis was in progress. The limits of the subperiosteal abscess beginning at the upper end are seen about half way down the shaft, but suppuration about the ankle in connection with the extension of the disease from the lower end of bone was beginning.

(By permission of Mr. WARD.)

immediate amputation was followed by a good recovery. In the specimen the medulla and cancellous tissue of the diaphysis are infiltrated with yellow foci of suppuration, and the outline of a large subperiosteal abscess is seen on the surface of the bone.

Ulceration of the bone has occurred in the usual place posteriorly (viz., at the juxta-epiphysial line), but is not extensive. The joint was not implicated.

The small amount of destructive bone change at the juxta-epiphysial region, coupled with the nature of the wide spread implication of the medullary tissue and the periosteum, justified the assumption that the poison must have been of an intensely septic character, and this was confirmed by Mr. Edmund Owen, by whom the specimen had been presented, and who in answer to an enquiry has written that the girl would have died of acute septic intoxication if amputation had not been performed.

The description of this case may be contrasted with the specimen illustrated in Fig. 9, in which the medulla has been spared in consequence of the slow progress that the suppurating process has been able to make.¹

3. NECROSIS.

On this subject there is little left to say that is not found in the text-books. Resulting always from subperiosteal abscess or osteomyelitis, it is open to question if it should not have been dealt with under those headings. Convenience, as well as precedent, however, justify its separate treatment.

Only a few points call for comment here.

(1). *Necrosis of compact tissue*.—Though in extreme cases the whole diaphysis or considerable portions of it may die, especially at that end at which the primary focus is situated, yet the form most commonly met with is necrosis of the compact tissue.

This form of necrosis is probably due to suppurative inflammation taking place in the vascular channels of the compact bone. Direct infection of the superficial layers of bone is very liable to

¹ Since this was written a second specimen of acute osteomyelitis of the central cavity has come under the writers' observation. It confirms much that has been said with reference to the specimen illustrated in Figs. 10 and 11.

A detailed description is appended to its photograph (Fig. 12).

occur when it is covered with pus under considerable tension. But necrosis, though a usual result of acute subperiosteal abscess, is not by any means a necessary one, and this fact shows that the interference with the blood-supply from a stripping up of the periosteum is not a sufficient explanation of it.

Necrosis rarely extends beyond the compact bone when the infection is from the surface.

Those large cavities filled with granulation tissue and containing eroded fragments or plates of compact bone, which are so commonly seen in sequestrotomy of a bone such as the tibia, would seem to suggest that the necrosis had involved a large portion of the shaft, and that the compact tissue by reason of its density, was the only portion that had not been absorbed.

In view, however, of the infrequency of suppurative inflammation of the central medulla, the more probable explanation is that the compact tissue alone perishes at the time of the formation of the subperiosteal abscess, but the cancellous tissue in its vicinity is gradually destroyed by the rarifying osteitis through which the separation of the sequestrum is brought about.

(2) *Migration of sequestra*.—If a sequestrum forms in the vicinity of the growing end of a long bone, it may in the course of years, if not removed by natural or artificial means, be carried, in consequence of the growth taking place at the epiphysial line, a considerable distance away from it.

Thus, Fig. 13 shows a strip of compact tissue which had undergone necrosis at the age of 2. It can be recognised as belonging to the lower portion of the early shaft, and when the leg was amputated ten years later, it was found quite two inches distant from the epiphysial disc.

(3) *The importance of removing sequestra when loose*.—The same case illustrates an exceptional danger that may result from the neglect to remove a loose portion of dead bone. The sequestrum in the course of years, and in consequence of its migration and its gradual action upon the enclosing bone, had come to lie almost transversely, transfixing the shaft of the femur. The end that protruded upon the inner side had ulcerated into the femoral

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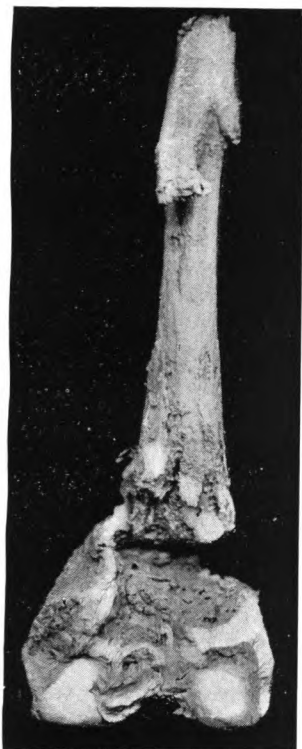


FIG. 14.

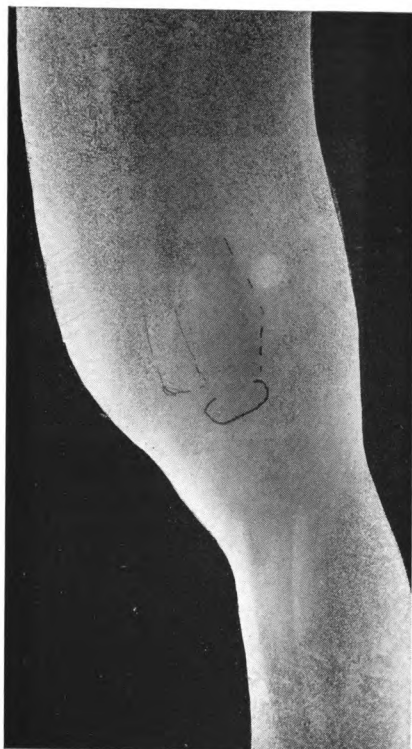


FIG. 15.

FIG. 14.—From a boy. Subperiosteal abscess, suppurative arthritis, separation of epiphysis. Amputation. Before amputation there was considerable deformity of the joint simulating sub-luxation, but in reality due to displacement backwards of the leg and the attached epiphysis of the femur. The lower end of the diaphysis lay bare in the suprapatellar pouch of the joint. The specimen shows that suppuration and destruction of bone has taken place over the whole juxta-epiphyseal area. The lower end of the diaphysis is much eroded and also the surface of the compact tissue for some distance. The limit of the subperiosteal abscess is evident.

FIG. 15.—From a child two years old. On incising a subperiosteal abscess, the lower epiphysis of the femur was found separated. There was no history of injury. Joint not involved. On re-admission for sequestrotomy, this skiagram was taken. It shows displacement backwards of the lower epiphysis. This is united to the new bone thrown out by the periosteum, which has retained its attachment to the epiphysis. The joint could be placed in the position of genu recurvatum.

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FIG. 13.

FIG. 13.—John W., æt. 11, had an abscess in the lower part of the thigh at the age of two, and ever since there had been sinuses. Admitted for hæmorrhage of a serious character from the sinuses. The limb was amputated. The specimen shows a considerable sequestrum, representing a vertical portion of the compact tissue of the early diaphysis. This lies obliquely and its ends project through apertures on either side of the femur. On the inner side it has ulcerated into the femoral artery, and the circle of fishing gut seen on the left side passes round a rod lying in the artery at the point where it is exposed by the hole in the vessel. In consequence of the growth that has taken place at the lower epiphysis the sequestrum has been displaced upwards for at least two inches and has also come to lie obliquely. The front of the bone has been chiselled away to expose the sequestrum.

artery, and led to severe recurring attacks of hæmorrhage, for which the limb was amputated.

4. SEPARATION OF AN EPIPHYSIS.

Separation of the epiphysis may result from suppuration extending transversely over the whole juxta-epiphysial area.

Such a condition is shown in Fig. 14, and similar specimens are not rare. Many of these cases end in amputation, but under favourable circumstances it is possible for an epiphysis separated in this way to unite. This actually occurred in the case from which the skiagram (Fig. 15) was taken, but as a sequestrum is still present, and the limb assumes the position of genu recurvatum, the patient cannot be regarded as having completely recovered.

5. ACUTE SEPTIC ARTHRITIS.

When this occurs as a consequence of inflammation of growing bone it is brought about in one of several ways.

(1). *From anatomical predisposition.*—In the case of certain joints their implication is very largely dependent upon the anatomical relations of the epiphyses forming part of them. Thus the hip-joint is almost certain to suppurate when the disease originates at the upper epiphysial line of the femur, because the head and a large part of the neck lie practically within the joint. And if the primary focus is situated at the ossifying edge of the cartilage forming the acetabulum (Fig. 22), it is impossible to conceive any other outlet for pus except into the articulation.

The elbow is another joint that may suffer in consequence of the position of the upper epiphysis of the ulna. This, after the tenth year, is only a small disc situated at the extremity of the olecranon process and the chief part of the great sigmoid fossa is fashioned from the upper end of the diaphysis, consequently the attachment of the deep layer of periosteum to the epiphysial line cannot, in the case of the ulna, act as an outlying barrier for the protection of the joint from the extension of a subperiosteal abscess. In the case from which Fig. 7 was taken, suppuration, which began at the lower epiphysial line after a separation of the epiphysis, stripped up the periosteum along the whole length

of the bone and found an entrance to the joint on the inner side of the insertion of the brachialis anticus tendon.

(2) *As a consequence of subperiosteal abscess of the epiphysis.*—Suppuration commencing as usual at the juxta-epiphysial line may lead to an acute abscess beneath the periosteum covering the epiphysis. This is necessarily so small as to be incapable of recognition clinically. The pus strips up first the periosteum and then the articular cartilage, and finally finds its way into the joint. It is not easy to understand how it finds its way under this portion of the periosteum, for according to ordinary anatomical arrangements it would be expected to find an outlet underneath that membrane where it covers the diaphysis. This it does in the very large majority of cases, but that it may take the unusual course here stated is shown by the specimen depicted in Fig. 16. Possibly it may find an easy passage along the course of one of the vessels which according to Poland² sometimes perforate the epiphysial disc, or possibly the conjugal cartilage may be softened by the inflammation and permit the passage of pus through it, as later on it will be shown to do under other circumstances.

(3) *From direct extension of the suppurative process through the epiphysis.*—Suppuration of a joint not infrequently depends upon the inability of the conjugal cartilage to withstand the softening effects of the acute inflammatory process going on just beneath it. It loses over a varying area its characteristic appearance, and in the specimen is found to be converted into a soft, washleather-like tissue, or even granulations.

By the destruction of a part of this barrier, the pus finds an entrance to the epiphysis proper, and eventually reaches the joint in one of two ways:—

(a) Either it tracks round the circumference of the ossified portion of the epiphysis, separating it from its as yet cartilaginous periphery, until it reaches the articular cartilage, and breaks through into the joint (Fig 17), or

(b) It may extend through the cancellous tissue of the epiphysis as an acute osteo-myelitis. (Fig. 9).

² Poland, Traumatic Separation of Epiphysis, p. 20.

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FIG. 16.

Fig. 16.—The head of the tibia from a boy aged about 13. The leg was amputated for profuse suppuration among the calf muscles following acute arthritis of the knee. There had been no subperiosteal abscess of the diaphysis, but the pus extending from the primary focus in the juxta-epiphysial region had emerged on the surface at the crack seen in the photograph below the patch of bare bone which corresponded with the situation of the insertion of the semimembranous tendon. It had found its way under the periosteum covering the epiphysis and had stripped up the articular cartilage before opening into the joint, leaving a considerable portion of the articular surface of bone bare.

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FIG. 17.

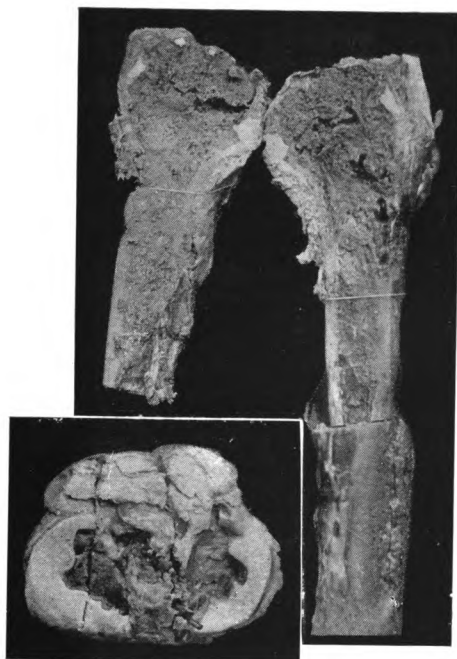


FIG. 18.

FIG. 17 and FIG. 18.—Photographs of the tibia from the amputated leg of a young boy. There had been acute arthritis of the knee, and subperiosteal abscess of the tibia.

Fig. 17.—*Antero-posterior section*.—Only the lateral terminations of the conjugal disc are seen, but in the specimen it could be recognised by a soft, wash-leathery line, except in some places where it had completely disappeared. On either side are marked changes. A large portion of the epiphysis is necrosed and is separated from its cartilaginous periphery over a very considerable area. This separation, by interfering with the blood-supply, is probably responsible for the necrosis. In the diaphysis the carious and necrotic changes are limited to the portion just below the epiphysal line, the greater extension on the right of the right hand specimen being due to the prolongation of the epiphysis downwards in the tubercle of the tibia. The subperiosteal abscess has arisen in connection with this part of the mischief. The suppurative process starting in the juxta-epiphysal region has softened and permeated the conjugal cartilage and found an easy path round the periphery of the bony epiphysis until it has reached the articular cartilage and opened into the joint. On the side of the diaphysis it has evidently extended more deeply into the bone, yet extension has taken place more rapidly laterally where the cancellous tissue is of more recent formation.

Fig. 18 shows the articular surface, and a glass rod indicates the position in which pus has found its way to the joint.

(By permission of Mr. TEALE.)

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FIG. 19.



FIG. 20.

FIG. 19 and FIG. 20.—The two illustrations show the cause of suppuration in both knee-joints of a boy *æt.* four weeks.

Fig. 19 shows an abscess in the cartilaginous epiphysis which contained a small cretaceous mass. This abscess had opened into the joint. The diaphyseal side of the cartilage is seen to be uninjured.

Fig. 20 shows an abscess in the juxta-epiphyseal area, which has hollowed out the diaphysis more than the cartilage. This also had opened into the joint. These photographs serve to illustrate the different positions in which the primary focus originates, viz., at the centre of ossification and at the juxta-epiphyseal region.

(By permission of Sir THOMAS SMITH).

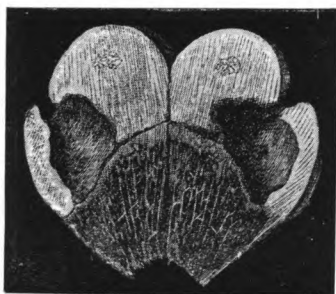


FIG. 21.



FIG. 22.

FIG. 21.—The head of the left femur of a congenital syphilitic infant, who had suppuration of three joints, and died at twelve months of age. This abscess "was situated partly in the ossifying cartilage and partly in cancellous bone," and opened on the neck of the femur, close to the digital fossa.

(By permission of Sir THOMAS SMITH, figs. 19, 20 and 21 are taken from his article on "Acute Arthritis of Infants," (Cases 2 and 3,) in *St. Bartholomew's Hospital Reports*, vol. 10, p. 192).

FIG. 22.—Olive —, *æt.* six months. Acute suppuration of the right hip-joint, erysipelas, purulent peritonitis. Death. An aperture was found in the acetabulum, leading through the articular cartilage to a small abscess cavity. In the photograph the acetabulum is seen divided across, so as to show the abscess. In the upper half a glass rod marks the track from the abscess cavity to the interior of the joint, and below it a bristle is inserted into the cavity itself. In the lower half a piece of glass rod has been laid laterally in the abscess cavity, which is situated between the articular cartilage and the bone. The abscess is close to the origin of the ligamentum teres. Below the glass rod is a pin.

In either case, necrosis of the epiphysis, more or less complete, results, and as the articular cartilage is usually largely destroyed, the necrosed surface lies exposed in the joint.

In these cases, a study of the specimens leads to the belief that the focus of the disease is situated near the central portion of the juxta-epiphysial region, and not near the periphery. The disease has usually led to considerable mischief below the epiphysial line, and has probably penetrated it and got well started in the epiphysis before it reaches the periphery, and leads to a subperiosteal abscess, which, in the majority of cases, even though the joint is affected, is the condition which is most obvious when the patient comes under surgical treatment.*

(4). *In consequence of the cartilaginous nature of the epiphysis in early infancy.* "*Acute Arthritis of Infants.*"—In very young infants, where the epiphyses are almost entirely formed of cartilage, the readiness with which the young cartilage disappears before the suppurative process gives a very different character to the disease. Abscesses develop in the soft cartilage whether the primary focus happens to be at the juxta-epiphysial line or in connection with an ossifying centre, and their cavities are frequently found to open directly into joints.

This condition has been carefully described by Sir Thomas Smith in his well-known paper on *Acute Arthritis of Infants*, (*St. Bart.'s Hospital Reports*, vol. x., p. 192), and the writer is indebted to him for permission to reproduce three photographs from that paper, which seem clearly to suggest the positions in which the mischief begins, and to illustrate the peculiar features which acute septic inflammation of growing bone assumes at a very early age in consequence of the difference in nature, and possibly of texture, of the tissues at that period (Figs. 19, 20, 21).

6. DEEP-SEATED ABSCESS.

This complication, apart from necrosis and periosteal abscess, does not seem to have received any attention in the text-books, although it cannot fail to be familiar to most surgeons.

* In a discussion on this paper, at the Leeds and West Riding Medico-Chirurgical Society, Mr. Littlewood referred to a case in which it was easily demonstrated that infection had reached the joint along the popliteus tendon.

The origin of acute abscesses beneath the deep fasciæ is often very obscure. Glandular or lymphatic suppurations probably account for many cases, but another large group undoubtedly originates in connection with growing bone. There are a considerable number of situations, apart from the epiphysial ends of the long bones, where, during childhood and youth, cartilage is being transformed into bone. The epiphysis of the great and small trochanters of the femur, and of the coracoid process of the scapula, the sacro-iliac synchondrosis, the cartilaginous bond of union between the rami of the pubes and ischium, and the small cancellous carpal and tarsal bones which possess centres of ossification, are a few that may be mentioned. The three first exemplify the fact that these subsidiary epiphyses often serve for the origin or insertion of powerful muscles, and are consequently likely to experience occasionally some of the ill effects of strain and tendon pull.

The small area covered by some of these epiphysis enables the pus quickly to find a way to the surface of the bone, and the periosteum is in many cases soon penetrated, possibly because it is thin or because the origins of muscles have rendered it firmly adherent. An abscess among the muscles results. There is frequently hardly any stripping of periosteum, and necrosis may or may not follow, and when it does it is often trivial. Consequently many of these acute abscesses clear up without trouble after incision, and unless the source of the mischief is found by careful search, their real character may be missed. Further, it will be seen from the cases that follow, that when inflammation of bone originates at one of these somewhat inaccessible growing spots it may prove a fruitful source of difficulty in diagnosis, or of error.

CASE 1.—A boy, aged 6, was admitted to the Fever Hospital for (?) typhoid, and in a few days was transferred to the Leeds General Infirmary for an acute abscess in the adductor region. The hip was unaffected. The abscess was opened and a small area of bone was found bare and rough at the junction of the rami of the pubes and ischium: a small transverse notch indicated where the cartilage had probably been eroded. The abscess healed quickly without any exfoliation of bone.

CASE 2.—A youth of 15 had been in the medical wards for two or three weeks with a high temperature, and pain about the right hip. The joint was evidently unaffected. No diagnosis was made. The development of a swelling in the right iliac fossa led to the writer being asked to see him. A fluctuating tumour could be felt, from the rectum, to lie over the sacro-iliac joint. The condition was obviously acute and not tubercular. A diagnosis of suppuration originating at the sacro-iliac joint in connection with growing bone was suggested. The case was transferred to Mr. Littlewood, who opened the abscess, and after a patient search found a small patch of bare eroded bone just at the edge of the synchondrosis. No necrosis resulted and the abscess healed without trouble.

CASE 3.—A boy of 12 developed a very acute abscess about the left shoulder two or three days after being pushed against by a schoolfellow. It was opened in the axilla and the finger passed between the two pectoral muscles to the coracoid process, which was quite bare. This process necrosed and came away before the resulting sinus healed.

CASE 4.—Specimen 1218 E in the Royal College of Surgeons Museum, is a scapula from a boy of 12, who died of pyæmia after a fall on the shoulder ten days before.

The root of the coracoid epiphysis is denuded of its periosteum, and the intervening cartilage is in great part destroyed. A subperiosteal abscess occupies the dorsal and ventral surfaces of the scapula, and these communicate freely above the neck of the bone. A full description of this case is given by Poland in *Traumatic Separation of the Epiphysis*, p. 160.

CASE 5.—An infant, two months old, was admitted with an acute abscess about one ankle. This was incised. The child subsequently developed erysipelas and died of convulsions. The cause of the abscess was necrosis of the centre of ossification of the astragalus. (Fig. 1.)

OTORRHEA AND MASTOID ABSCESS.

Before this portion of the subject is dismissed, attention may be directed to suppuration arising in connection with the temporal bone.

"Mastoid abscess" is generally looked upon as secondary to a chronic aural discharge, resulting from an otitis media. It is admitted that an otorrhœa and a mastoid abscess may be due to a primary disease of the bone, but so far as the writer can learn, this primary disease has usually been regarded as tuberculous. It is probable, however, that not a few of these primary bone inflammations resulting in abscess are, in their pathology, identical with the acute periosteal abscesses which arise in connection with the inflammation of growing bone. From this point of view the development of the temporal bone is of interest.

Of its three portions the squamosal and the tympanic are formed in membrane, and it may be noted in passing that this acute infective disease rarely, if ever, attacks these or any other of the membranous bones. The petrosal, from which the mastoid is subsequently developed, arises by four centres in cartilage. The growth of these bony centres is so rapid during the later months of foetal life, that at birth all that remains of the cartilage is a thin intervening plate at the squamo-petrosal suture, and this becomes completely ossified during the first year. (Fig. 23 shows the squamo-petrosal suture). So long as it exists it is, in effect, an epiphysial disc where growth is taking place, and is no doubt as prone to be attacked by suppurative organisms under favouring conditions as conjugal cartilages elsewhere.

Fig. 24 shows well the ridge on the squamosal at birth which marks the squamo-petrosal suture and the position of the conjugal cartilage. It lies immediately over the tympanum, and any abscess originating in connection with growing bone at the uniting cartilage would be almost certain to find its way into the upper part of the tympanum. Not only would gravity favour this, but the difference in tension between the middle ear and the intracranial contents, as well as the firmer attachment of the dura mater on the upper surface of the bone, would no doubt tend in the same direction.

The following case, which Mr. Nunneley has permitted the writer to make use of, and from which the specimen shown in Fig. 23 was prepared by Mr. Secker Walker, is very probably an instance of this mode of origin of a mastoid abscess. Like

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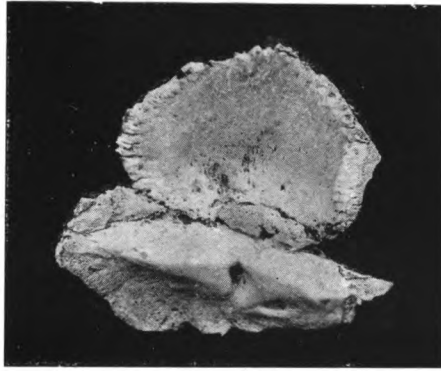


FIG. 23.

FIG. 23.—The temporal bone of W. H. B., æt. three months, shows the line left after suppuration and maceration have removed the cartilage at the petro-squamosal suture. A portion of the squamous bone has been broken in preparation.

(By permission of Mr. SECKER WALKER).



FIG. 24.

FIG. 24.—The squamosal portion of the temporal bone at birth. The ridge which unites with the petrous portion at the petro-squamosal suture is well shown, and the situation of the conjugal cartilage with respect to the middle ear can be readily appreciated.

(Figs. 23 & 24 are from specimens prepared by Mr. SECKER WALKER).

many cases of otorrhœa supposed to be due to primary disease of bone, it occurred in the first year of life before the petro-squamosal cartilage had disappeared.

W. H. B., aged 3 months, had a purulent discharge from the left ear for some time before admission, and facial palsy supervened. There had been no exanthem or other serious illness except occasional convulsions before the aural discharge. The mastoid process was found soft and carious and the antrum filled with soft caseating material. The child lived a week after the operation, during which there was general rigidity but no convulsions.

Post-mortem.—A few tubercles upon the surface of the spleen. No tubercle in the lungs. *Brain.*—Increased arachnoid fluid, but no distension of ventricles; the left half of the cerebellum was softer than the right and easily broke down.

The condition in the left half of the cerebellum, the absence of tubercular meningitis, and the specimen itself, are all more in favour of an acute infective inflammation of growing bone than of tubercle. The notes suggest that tubercle was the dominant idea throughout the case and possibly explain the description of the spleen.

A specimen, belonging to Mr. Arthur H. Cheatle (No. 618, Catalogue of the Otological Museum—6th International Otological Congress, 1899) is also suggestive.

It is the "left temporal bone of a child aged one year who died of suppurative meningitis. Membrane intact and bulging—middle ear full of muco-pus containing streptococci and staphylococci; lining membrane thick and congested looking; veins from middle ear to the petro-squamosal sinus marked—roof of middle ear was intact."

APPENDIX.

To prevent confusion, certain unusual and interesting cases are dealt with here, apart from the main plan of this article, whilst some other conditions are alluded to chiefly for the sake of comparison.

TUBULAR OR PYRAMIDAL ABSCESS OF BONE.

There is a very unusual form of abscess in bone whose mode of origin is somewhat doubtful. The abscess is tubular or pyramidal in shape, with its base resting upon a line corresponding to the situation of the conjugal cartilage, and its apex directed towards the medullary cavity. It runs a chronic course.

In the two specimens referred to below, the abscess was situated in the lower part of the diaphysial portion of the femur, and in both a sequestrum was or had been present. The specimens suggest that a suppurating inflammation of bone, resulting in necrosis, has occurred in the neighbourhood of the conjugal cartilage, and that the abscess has been walled up by a sclerosing osteitis around it, extension taking place directly upwards towards the medulla, probably because the density of the bone in this direction was least.

But the difficulty of accounting for suppuration taking place in this situation arises from the ages of the patients.

In both the conjugal cartilage should have been completely ossified years before the first signs of trouble showed themselves, and yet the position and peculiarities of the abscesses give rise to a strong impression that they originated from a portion of the epiphysial cartilage that had failed to disappear.

Fig. 25 is a photograph of a specimen which was taken from a leg amputated by Mr. Mayo Robson. In the lower part of the abscess cavity there was found a small sequestrum lying vertically, and since lost.

The patient was a woman of 35, in whom the first signs of the disease had appeared five years before.

No. 1247 G in the Royal College of Surgeons' Museum is a very similar specimen from a man of 46, whose illness began nine years before amputation. A sequestrum was removed.

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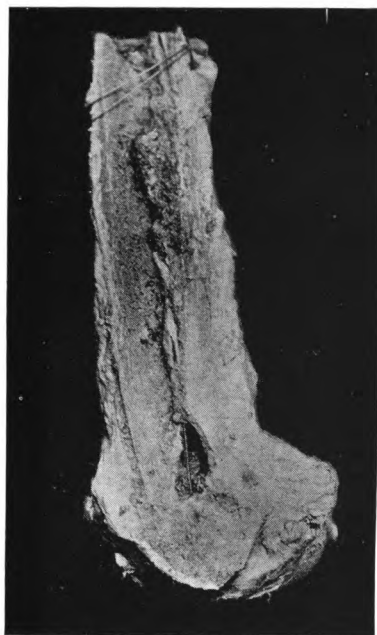


FIG. 25.

FIG. 25.—Tubular or pyramidal abscess of bone. The upper part of the abscess sac has been laid open to show the glass rod, which is better seen in the basal portion of the cavity.

(By permission of Mr. MAYO ROBSON).

**TWO-FOLD INFECTION.—ACUTE INFECTIVE INFLAMMATION OF
BONE ASSOCIATED WITH TUBERCULOUS SYNOVITIS.**

The following case is of considerable interest :—

J. H. G., a boy, *æt.* 11, whilst playing cricket was seized with sudden pain in the right knee. Till that moment he had been quite well. The next day he had to lay up, and in due course an abscess formed, which at the end of three weeks was incised, and a pint of pus let out. Ten months later he was admitted to the Leeds General Infirmary. The knee had all the characteristics of a tuberculous knee-joint, which had gone on to subluxation. There was also a good deal of thickening about the lower end of the femur, and a sinus opened on the outer side. Excision of the knee was performed. The joint was in that stage of pulpy disease in which the condition was going on to repair. The granulation tissue was largely transformed into fibrous tissue, and the patella was ankylosed to the femur. In the lower part of the femoral diaphysis a large abscess cavity was found containing a sequestrum of the compact tissue two inches long by one inch broad. This cavity communicated by the sinus with the exterior, but was completely separated from the sawn surface of the epiphysis by the healthy conjugal cartilage. Good union took place.

Here, in close proximity, there existed two distinct diseases—a suppurative inflammation of growing bone speedily resulting in abscess and necrosis, above the epiphysial line, and a tuberculous and non-suppurative inflammation of the synovial membrane of the knee-joint, involving the lower epiphysis of the femur, below it.

The history of the onset of the former was readily forthcoming, but the tuberculous state had come on gradually and unnoticed. All that the patient or his friends could say was that the knee had been quite well and like the other before the acute condition set in.

It may be presumed, therefore, that the two diseases originated at the same time, or that the tuberculous state became grafted

upon a simple inflammatory synovitis, induced by the acute suppuration in the vicinity of the joint.

NECROSIS IN THE FULLY OSSIFIED LONG BONES.

Necrosis which is neither traumatic, tuberculous, nor syphilitic, is occasionally met with as a result of an apparently spontaneous acute inflammation affecting a portion of a long bone.

Such cases are rare, and whatever their true pathology may be, it is clear the inflammation does not originate where cartilage is being transformed into bone. The inflammatory focus is, on the other hand, probably a considerable distance from the vicinity of an epiphysis.

The following case illustrates this and forms an excellent contrast to the conditions so frequently occurring in children and young adolescents.

W. B., æt. 43, a robust man, had been out on strike six weeks when his right forearm began to swell for several inches above the wrist. In a few days it was incised, and it continued to discharge for three years, when the writer removed two sequestra—one of compact bone, and the other of cancellous tissue as big as a marble—from a cavity surrounded by sclerosed bone. This cavity was quite one and a-half inches from the wrist, and a considerable distance from the ordinary level of the epiphysial line. No history of injury or of probable tubercle could be obtained, nor had the patient suffered from want of food when on strike.

THE IMMUNITY OF MEMBRANE-BONES.

One exceedingly interesting point in connection with the subject of this paper is the immunity which bones developed from membrane possess in regard to acute infective suppuration.

The writer has never seen an instance in which suppuration or necrosis about the skull or face or lower jaw could be attributed to the acute infective suppurative disease of bone which is so very common in bones growing from cartilage before ossification is complete; nor has he been able to find in three museums (Hunterian, Guy's, and Leeds) a single specimen of cranial necrosis which could with any probability be referred to that disease.

*On Acute Septic Inflammation of Young Bone
Growing from Cartilage.*



FIG. 26.

FIG. 26.—Matthew W., æt. 26. Chronic suppurative osteomyelitis of the whole frontal bone ; showing his present condition.

The most likely explanation of this immunity is the absence of a conjugal disc, and of the weak and vulnerable juxta-epiphysial region. In the long bones the epiphysial disc and the juxta-epiphysial region introduce not only a place of least resistance but certain anatomical peculiarities, which are of considerable importance in determining the various complications of infective suppuration. This predisposing factor is absent in the membrane bones and with it the typical complications by which the disease is readily recognised.

Necrosis, other than syphilitic, is usually the result of direct injury, but the bone is generally in contact with a septic wound, and the inflammatory process which leads to its death is doubtless secondary, and is speedily limited.

Osteo-myelitis is also met with, for portions of the whole thickness of a bone will necrose. Yet osteo-myelitis is rarely extensive and is a part of the general osteitis associated with the septic wound. The following case of an osteo-myelitis which involved the whole diploe of the frontal bone, might at first sight very possibly be thought to be an instance of the acute infective variety, but the gradual way in which by successive extensions, once connected with fresh injury, the condition spread during a period of years, makes it present a very different clinical picture to the acute osteo-myelitis which leads to necrosis of the diaphysis in the long bones. In this instance there was no necrosis of the inner or outer table but only of the diploe.

Matthew W., æt. 26, ten or twelve years before his final admission to the Leeds General Infirmary in 1898, received a scalp wound over the left eye, which was stitched up. Some weeks later he came under Mr. R. N. Hartley, at the Leeds Public Dispensary, for a tender swelling at the outer side of the orbit. The outer plate of the frontal bone was gouged through, and a small area of diseased cancellous bone was scraped away. This relieved his pain, but the disease continued to extend and reached the situation of the frontal sinuses. At intervals four more similar operations were performed, including drainage of the frontal sinuses.

Sinuses and offensive discharge persisted, and in 1896 he was kicked on the head by a horse, and admitted under Mr. Robson

into the Infirmary. From this time onward the writer had opportunities of seeing the patient when he made occasional reappearances, and his condition became more and more distressing in consequence of the amount and foul odour of the discharge. In January, 1898, he removed, at two operations, a large horse-shoe of the outer table. This table was half an inch thick and very dense, and between it and the inner table a quantity of foetid granulation tissue, containing innumerable small cancellous sequestra, was found. After this had all been cleaned away, the cavity between the two plates was found to extend over the whole frontal bone. The patient made a satisfactory recovery, and is now quite well, except that the prolongation of the frontal sinus over the right orbit has not yet completely filled up. (Fig. 26).

SOME NOTES ON THE ÆTIOLOGY OF STRABISMUS.

By ARTHUR W. ORMOND, F.R.C.S.

ASSISTANT SURGEON, ROYAL EYE HOSPITAL, SOUTHWARK.

OPHTHALMIC REGISTRAR AND TUTOR, GUY'S HOSPITAL.

THE Ætiology of Strabismus is a matter over which ophthalmologists have long contended, and one which even now has many points still unproved. There are, however, cases, perhaps the majority, that may be satisfactorily explained, and it is my object in this paper to record the notes of a selected number of these cases, taken mainly from the out-patient department of Guy's Hospital, and compare the various theories explaining strabismus with the actual facts.

By strabismus, or squint, I mean the condition in which the visual axis of one eye deviates from the visual axis of the other, the axes not meeting at the object looked at, the angle of deviation remaining the same, in whichever position the eyes are turned. This excludes all those cases of deviation of the visual axis of one eye from that of the other, due to paralysis or paresis of one of the extrinsic muscles of the eye.

Strabismus manifests itself early in life, the patient squints if he or she has the conditions necessary for its manifestation in the majority of cases from the third to the fifth year of life,

but a concomitant squint may appear at any age, since, as I shall point out later, a latent squint may become manifest if the visual acuity of one eye should become seriously depressed below that of the other; but convergent concomitant squints generally manifest themselves before the tenth year. Consequently we have to look for the cause of squints among conditions existent in the eye during the early months of life.

According to Fleischsig, the formation of medullary sheaths to the fibres of the optic nerves has commenced at birth, but has not extended centripetally to the cortical area, consequently we may assume that at birth there is no isolated conduction along the fibres of the optic nerve to the brain.

Further, in order to correctly appreciate the appearance of objects in space, as to form, colour, etc., the centres of the other sensory organs must be developed and the transcortical fibres connecting these with the visual centres must be also developed. Probably a period of some two or three years is passed during childhood before the centres and their communications one with another are fully matured.

Baumgarten asserted some years ago, and experience confirms his observation, that a newly-born infant saw merely light, and that only quantitatively. We all know the vacant stare of very young children, the inability to fix the eyes for more than the shortest time, and the isolated movements of one eye and often of one lid.

In the Philosophical Transactions of 1728, Cheselden reports a case of a young man receiving sight after couching of the cataract in each eye, he previously having been able only to see light and discern colour in mass. In this case, and in others that have been published since, we have the statement of intelligent adults on the gradual development of vision in their own eyes.

“ When he first saw he was so far from making any judgment about distances that he thought all objects whatever touched his eyes (as he expressed it) as what he felt did his skin, and thought no objects so agreeable as those which were smooth and regular, he knew not the shape of anything nor any one thing

from another, however different in shape and magnitude. Upon being told what things were, whose form he before knew from feeling, he would carefully observe, that he might know them again, but having too many objects to learn at once he forgot many of them, and (as he said) at first he learned to know, and again forgot a thousand things in a day. One particular only (tho' it may appear trifling) I will relate. Having often forgot which was the cat and which the dog, he was ashamed to ask, but catching the cat (which he knew by feeling) he was observed to look at her steadfastly, and then setting her down said, 'So, puss, I shall know you another time.' He was much surprised that those things which he had liked best did not appear most agreeable to his eyes, expecting those persons would appear most beautiful that he loved most, and such things to be most agreeable to his sight that were so to his taste. We thought he soon knew what pictures represented, which were shewed to him, but we found afterwards we were mistaken, for about two months after he was couched, he discovered at once they represented solid bodies, when to that time he considered them only as party-coloured planes, or surfaces, diversified with variety of paint, but even then he was no less surprised, expecting the pictures would feel like the things they represented, and was amazed when he found those parts, which by their light and shadow appeared now round and uneven, felt only flat like the rest, and asked which was the lying sense, feeling or seeing.

At first he could bear but very little light, and the things he saw he thought extremely large, but upon seeing things larger, those first seen he conceived less, never being able to imagine any lines beyond the bounds he saw, the room he was in he said he knew to be but part of the house, yet he could not conceive that the whole house could look bigger. Before he was couched he expected little advantage from seeing worth undergoing an operation for, except reading and writing, for he said he thought he could have no more pleasure in walking abroad than he had in the garden, which he could do safely and readily. And even blindness, he observed, had this advantage, that he could go anywhere in the dark much better than those who can see, and

after he had seen he did not soon lose this quality, nor desire a light to go about the house in the night. And now being lately couched of his other eye, and looking upon the same object with both eyes, he thought it looked about twice as large as with the first couched eye alone, but not double that we can anyways discover."

The ability to use both eyes together is peculiar to man and some of the higher vertebrates, but in no animal is the function of binocular vision so highly developed and so essential as in man, a function by no means present at birth, but acquired by subsequent development. The appreciation of colour also is not demonstrable before the sixth month, and that of different colours is obtained by a gradual development, those nearer the red end of the spectrum being appreciated first.

In view of these facts we must consider the visual power as being gradually developed, the newly-born child starting with an organ possessing only the potential faculty of perfect vision, and not more power probably than mere perception of light. This is not strange, for we know that animals living in the dark have only rudimentary organs of vision, consequently we should be surprised if the newly-born infant possessed the power of seeing and discriminating before the brain and the other sensory organs were in a position to assist the eye, to an intelligent appreciation and discrimination of what was seen.

Donders' theory that squint was due to an abnormal relation of the power of convergence and accommodation, is not sufficient to explain all the cases of squint that present themselves to us, and the theory of amblyopia ex anopsia is quite untenable in the face of many of the facts which are at hand to-day.

Donders points out that hypermetropes of moderate degree have, in order to focus an object clearly, to use their accommodation in order to correct the static refraction of their eyes.

In the normal condition of an emmetropic eye a certain amount of accommodation entails a certain amount of convergence, and we generally say that an emmetrope accommodating one diopter converges one metre angle, two diopters two metre angles x diopters x metre angles; consequently a hypermetrope

with a defective static refraction cannot conform to this condition but accommodates in excess of his convergence, or converges in excess of the proximity of the image looked at. Hence one of two things must happen. The accommodation being used to bring the object into focus clearly, the visual axes of the two eyes do not meet at the object, but between the object and the observer, the image of the object falling on non-corresponding points of the two retinæ, diplopia or double vision ensuing. The other alternative is single but misty vision. In myopia the converse is the case, the accommodation being weak or unnecessary. Convergence is in excess and an effort to relax the convergence produces a tendency to divergence and eventually divergent squint. Although it is unsafe to argue that the relation of convergence to accommodation is the same in ametropia as in emmetropia, still this theory of Donders' is no doubt true in a large number of cases but it is not sufficiently explicit.

A choice has therefore to be made between double sight and misty sight, that is to say, if the visual axes fall on the object the image will be out of focus ; if, on the other hand, the object be focussed accurately double vision must ensue.

On which of these alternatives the choice will fall depends on the condition of the eyes. If, owing to any cause, such as a high degree of ametropia, opacity of the refractive media, etc., clearness of vision is impossible, then no squint will develop, the patient giving up clear vision in order to preserve single vision ; on the other hand, if the object can be seen clearly, then clear vision is preferred and the diplopia overcome, according to Donders, by an active suppression of the image in the squinting eye.

We undoubtedly have power to neglect the image in one eye. In gazing through a telescope, or an ophthalmoscope, or better still a microscope, with both eyes open, many people have the power of entirely neglecting the retinal picture in one eye. The images are of course absolutely different, and no effort is made to fuse them. By an effort, rendered easier by moving the microscope slide, we can perceive one image only.

We do not apprehend all we see, that is to say, a very small part of what we see, the image of which falls on the retina, ever reaches the state of consciousness, consequently it does not seem to me a matter of very great surprise, that in order to avoid a most annoying and muddling diplopia, a child should learn to neglect the image falling on one retina entirely.

But now arises an interesting question, Can this suppression of an image in consciousness lead to a condition of amblyopia such that the patient cannot, even by an effort, with the good eye covered, command good vision?

I believe, that if the amblyopia were due solely or mainly to an active suppression, that the patient would be able to command full vision again; but that we are in reality dealing with an amblyopia which depends for its existence upon some cause apart from the squint, and existing previous to the squint, so that the amblyopia is the cause of the squint rather than the squint the cause of the amblyopia. The condition of the amblyopic eye should be considered carefully in relation to this point.

When looked at from outside, or ophthalmoscopically, the amblyopic eye is apparently healthy. Whatever change is present is beyond the power of inspection or ophthalmoscopic examination to reveal.

The majority of cases show that one eye alone is involved, the other eye showing, as a rule, no evidence of a similar defect of visual acuity. The shape of the cornea, or refractive power of the media is different from that of the other eye, both eyes having an error of refraction, the higher error is usually in the amblyopic eye.

I have found cases in which the refractive error by retinoscopic examination was equal in the two eyes, but I do not remember seeing a case where the amblyopic eye had the lower error of refraction.

We may find that correcting glasses improve the visual acuity somewhat, or we may find that no improvement at all takes place with any glass; but if improvement does take place it is at the most partial only.

Colour vision is often unaffected, and although exceptions are found, the field of vision is generally complete; sometimes indeed scotomata, central or peripheral, may be demonstrated.

The presence of indirect vision renders the eye of great service to the possessor, even although the visual acuity may be less than one-sixtieth of the normal standard.

On questioning the patient, if old enough and sufficiently intelligent to understand, one is told that objects appear unreal, shadowy, blurred, or sometimes unsteady, as if they were vibrating.

The degree of amblyopia is not proportional by any means either to the length of time that the squint has lasted, or to the degree of error of refraction present.

The effect upon the squinting and amblyopic eye of covering the sound, non-squinting eye, for a prolonged period may be nil, or, on the other hand, may lead to a slow but real improvement, but not, however, to complete restoration of full acuteness of vision. Such an improvement is doubtless due to certain additional factors influencing the vision of the amblyopic eye. Since the eye has not been used for fixation, binocular vision being defective or absent, no effort to focus objects accurately has been made; and further, the object looked at having been seen indistinctly, we should expect a certain awkwardness to exist on first employing functions which have been for a longer or shorter time in abeyance—a sort of left-handed condition noticed in right-handed people, when owing to some injury the right hand is placed *hors de combat*. The improvement noticed in squinting eyes, on covering the sound eye for long periods is probably due to this awkwardness being gradually overcome.

If the amblyopia of the squinting eye were due to the effect of the suppression of the image of that eye we should expect the prolonged covering of an eye would lead to defective visual acuteness, and that in those cases where the eye has been bandaged for a long time, or owing to persistent blepharospasm the lids have been kept closed, that a permanent defect of vision would ensue, but this is only found to be the case where the affection ensued before full and complete development of the visual acuity had taken place. In adults this is not the case, since cases

of cataract developing during childhood obtain full vision after operation where the cataracts have existed for several years.

On the other hand, one can quite understand that any interference with the passage of light to the retina may have an effect on the proper development of the macula, but this interference must exist at birth or very shortly after ; the macula being once normally and completely developed is not permanently depressed by the exclusion of light.

"The excitation of light," says Dr. Steffan, "is sufficient to develop the functional activity of the eye ; under its influence the formation of the medullary sheath gradually extends in the optic nerve from the periphery, and its forcing influence is such that formation of the medullary sheaths takes place so rapidly in a child born in the eighth foetal month that at the end of a month the process is further advanced than it is in a child born at full term."

We see, therefore, that we have in the case of amblyopia in a squinting eye a condition presenting no visible change to the naked eye, a subjective rather than an objective change, accompanied, however, by many associated conditions, such as abnormalities in the shape of the eye or in the condition of the refractive media, sometimes areas of blindness in the field of vision, and always by a defect of acuteness of vision of a varying but unalterable degree. This defect has no relation to the duration of the squint, or to the age of the patient. Further, the same condition is not uncommonly found in cases in which there is not, and has never been, any squint. For these reasons it is impossible to believe that the amblyopia of the squinting eye is due to the squint.

We have, then, to ask ourselves the question, What is the cause of this amblyopia ? and that question cannot at present be satisfactorily answered.

As I have already pointed out, the non-squinting eye does not participate in the defect incident to its fellow. The non-squinting eye has full visual acuity, full field of vision with normal colour and light sense. Hence the lesion must be in front of the chiasma, since no lesion behind that point would give us clinically defective vision confined to one eye only,

without ophthalmoscopic signs. Any theory advanced must explain the main facts found in association with this amblyopia, viz., the unilateral lesion, absence of ophthalmoscopic signs, the higher error of refraction, the normal colour sense and field of vision, the fixed degree of amblyopia, the existence of the same kind of amblyopia without squint, and its occurrence at an early stage of life.

Vision in the new-born being limited, as already expressed, to quantitative perception of light, a very slight cause affecting the vision of one eye may lead to the whole of the effort to cerebrate being confined to one eye. In other words, that the image in one eye being better than that of the other, the child develops and unconsciously cerebrates the image formed on that retina only. The amblyopic eye then develops normally, but the image is not received into consciousness except when the retina receives impressions not received by the good eye, that is, in the field of vision peripherally. The function of binocular vision, then, is either developed imperfectly or not at all, and as the extent to which attention is paid to the image of either eye varies, so conversely would the amount of amblyopia. The non-development of the power to cerebrate the image in one eye as fully as that in the other, leads to the loss of power to do so even at will, with only one retina in use. There is no evidence that any actual structural defect exists, since microscopical examination of an amblyopic eye must certainly be rare, as such eyes are seldom, if ever, removed, except for other reasons, accidents, etc.

Consequently, any cause acting in the first months of life, and rendering the image in one eye less perfect than the other may lead to amblyopia of that eye.

Schleich examined one hundred and fifty newly-born infants and found in forty-nine cases (seventy-eight eyes) retinal hæmorrhages besides a few other changes.

Naumoff (*Archiv. of Ophthalm.*, xxxvi., 3 p. 180), in 1890, examined forty-seven infants at full term and found pathological changes in twelve pairs, due he considered to increase of intracranial pressure during labour.

Hæmorrhages of this nature would be absorbed and leave no trace behind.

The presence of a degree of astigmatism higher in one eye than another, due to a badly-shaped cornea or globe, early inflammation, blepharospasm, ulcers, or injuries, may depress the visual acuity for a longer or shorter time during the critical period of the development of the vision and so lead to amblyopia.

This theory, then, would explain the unilateral change, and also another fact not often noticed, viz., that the good eye, in spite of comparatively big errors of refraction develops more than usually acute vision, six-fifths being read quickly and easily, due of course to the undivided effort to appreciate the image in that eye; further, the existence of higher errors of refraction in the amblyopic eye, the absence of ophthalmoscopic signs, the presence of normal colour sense, the absence of any improvement by treatment and the occurrence at an early period of life.

Whether strabismus will develop or not depends on the existence of other factors which may be present; such as defects of accommodation or convergence, muscular abnormalities, etc.

We find strabismus present in patients who have no amblyopia; who possess eyes with some considerable, or, on the other hand, some slight error of refraction, but with good visual acuity in each, and we must consider why these patients squint, knowing as we do that the majority of patients with errors of refraction and good visual acuity in each eye do not squint. In these cases we have undoubtedly another factor of normal vision either wanting or defective, viz., the power of binocular vision or the power of receiving in consciousness the image in each eye in such a manner that the object looked at is seen by both eyes, and the resultant perception is due to a fusion of both images in consciousness.

By the arrangement of the optic nerve fibres, all objects seen on our right side are perceived by the left cerebral hemisphere, consequently binocular vision is possible in those cases only where complete decussation of the fibres does not take place.

We find in fishes that the two optic nerves cross entirely to the opposite side, no fibres remaining on the same side. The same is true of amphibia and birds, but as we pass higher in the scale we find that a larger and larger number of fibres do not decussate, and finally in man the eyes are placed in the frontal plane and about two-fifths of the fibres do not cross; that proportion, then, of the field of vision is therefore involved in binocular vision, the extent to which decussation does not take place, denoting the degree of binocular vision possible.

This function of binocular vision is not present at birth, but the conditions for its development being present, the function is acquired during early life.

Undoubtedly in a large number of squints binocular vision is either very imperfect or absent. By imperfect I mean that by displacing the image of one eye above that of the other diplopia is not obtained or obtained with difficulty. Often further efforts must be made to separate the images, as by putting a red glass in front of one eye and keeping the prism moving in front of the other before the two images can be recognised. If the visual acuity of one eye is much below that of the other, binocular vision will, of course, be bad, but one finds in certain cases defective binocular vision with good visual acuity in each eye. By means of this binocular or stereoscopic vision (as well as by other means) we appreciate the third dimension. If, then, this power in a certain case is not present there is a very much lessened stimulus for the two eyes to work together in a sort of double harness, but each will work irrespective of the other, and it is quite clear that no diplopia will be complained of.

Further, in the treatment of squint, the presence or absence of binocular vision materially affects the prognosis. If present, the chance of good recovery is infinitely greater than if absent. We also find, in some cases, that binocular vision is not present over the whole field, but only over a very small portion, and this area may, by proper means, be considerably extended. Binocular vision may be possible at a distance, but owing to the presence of a divergent squint, associated with defective convergence, on approximating the object looked at, is

not obtained, and *vice versa*. The function may be entirely absent, possibly due to a non-development of the power from birth.

In some cases of squint in children diplopia is complained of, and in these cases binocular vision is, of course, present; but the absence or diminution of this function explains the fact that diplopia is so seldom recognised.

Undoubtedly, as Donders first pointed out, ametropia is present in cases of strabismus in the vast majority of cases, and when ametropia exists, the usual relationship of convergence to accommodation is altered; for it is not accurate to argue that the relation between accommodation and convergence which is developed in emmetropia in consequence of daily practice exists for all conditions of refraction. That the ciliary muscle is affected by the refraction is well known, a larger development of fibres taking place in hypermetropia than in emmetropia, and a smaller number in myopia. Besides, a far larger number of cases with errors of refraction do not squint, and taking cases of squint alone, we do not find that even here the majority show errors of refraction only, the two eyes being normal in every other respect. Certainly, a fair number of cases, where the relationship of accommodation and convergence satisfactorily account for the squint exist, but other causes are more frequent.

Great defects of converging power in cases of emmetropia or small degrees of astigmatism, are sometimes found among our out-patients where not the slightest movement is made by the visual axes to converge when an object is approximated to the eyes, one or other eye fixing, whilst the other diverges. Binocular vision in these cases is, of course, defective, since the two eyes can never be directed towards an object close to them, but only when the object is far off.

The lesion in these cases is probably nuclear, and binocular fixation being impossible for most of the functions of life, binocular vision is weak and generally divergent squint develops.

This defect leads us to another undoubted cause, the existence previously of latent squints. Many of those cases of squint noticed in cases where for some reason blindness or severe

impairment of vision has occurred in one or both eyes, are due to this cause. The tendency for divergence to occur, as adolescence is reached, accounts for the spontaneous cure of some cases of convergent squint, and also cases where tenotomy having been performed early in life and parallelism maintained for some time, subsequent divergence ensues.

The majority of latent squints are due to insufficiency of the internal recti, but a preponderance of these same muscles may also occur and a latent convergent squint exist. The reason these squints do not become manifest, is because good binocular vision exists and a most annoying diplopia would result if a constant effort did not keep the visual axes directed, so that the two images are superimposed, and one image received into consciousness; if, however, the image in one eye is displaced vertically, then the diplopia is readily recognised. Consequently, if from any cause this objectionable diplopia ceases to exist, owing to the acuteness of vision being severely diminished in one or both eyes, then the eyes will take up the position of equilibrium due to the muscular defect and a convergent or divergent squint ensues, as the case may be.

The notes of a case where fusion was impossible owing to the existence of an altitudinal squint in addition to divergence are recorded lower down.

Paralysis or weakness of accommodation may also possibly lead to a development of squint, owing to the absence of the stimulus to convergence that the accommodative act usually involves. The frequency with which some illness precedes the manifestation of squint in children lends some colour to this idea, since the intrinsic muscles of the eye may suffer in the general debility during convalescence following one of the exanthemata. A case of divergent squint with diplopia, in a boy of eleven, following a severe illness, has recently come under my notice. This patient has complete paralysis of accommodation.

In conclusion. Strabismus of the concomitant kind is due in the majority of cases to absent or defective binocular vision. The greater number of cases have defective binocular vision because the visual acuity of one eye is very defective. This defect

again is in a large number of cases due to amblyopia. No satisfactory explanation of this amblyopia is at present known to us.

The view that appears therefore to be most satisfactory is, that in consideration of the condition of the eyes, as regards vision at birth, a very slight interference with one eye may lead to a deterioration of the image formed in that eye. This having occurred, the better image alone is appreciated and developed into consciousness, the other eye only being used to extend the field of vision, and never being appreciated in consciousness to the same degree. This function being undeveloped during the developmental period is permanently lost. The degree of amblyopia depends on the extent to which the image of the eye has been received into consciousness, that is, the extent of its contribution to the single image seen binocularly.

Other causes leading to defect of visual acuity of one eye, lead also to defective binocular vision, such as opacities of the media, fundus changes, anisometropia, etc.

Other causes of squint are numerous, but errors of refraction, abnormalities of the extrinsic muscles, or of the power of convergence or accommodation, will explain most of the cases remaining.

CASES OF SQUINT SELECTED FROM THE OUT-PATIENTS OF GUY'S
HOSPITAL DURING THE LAST TWO YEARS.

Cases where the squint was primarily due to impaired binocular vision.

A. B., *æt.* 18.—Convergent concomitant strabismus noticed for many years. Movement of eyes good, laterally, can fix with either eye. Has no binocular vision. Refraction under atropine.

$$\begin{array}{c|c} +1.5 & \\ \hline & +2.5 \end{array} \qquad \begin{array}{c|c} +2.5 & \\ \hline & +2.5 \end{array}$$

With correction vision is $\frac{5}{6}$ in both eyes.

E. T., *æt.* 15.—When this patient gazes steadily in the distance, the right eye is noticed to diverge, the action being recognised by the patient, she stating that she knows some change has taken place.

Binocular vision obtained only with difficulty by means of prism and red glass; either eye will diverge when the other is covered.

Converging power good

Refraction under atropine B.E.

$$\begin{array}{c|c} +1.5 & \\ \hline & +1.5 \end{array}$$

V.E.E. = $\frac{5}{6}$

N. Y., *æt.* 15.—Convergent concomitant squint, angle about 45° . Can fix with either eye. Convergence good. Vision with glasses $\frac{5}{6}$ with each eye. Has no binocular vision. Low degree of hypermetropic astigmatism.

Cases due to defective binocular vision associated with large errors of refraction.

L. F. B., *æt.* 23.—Had convergent concomitant squint when a child, and for this was operated on. When examined this year some divergence was present.

Binocular vision obtained with difficulty.

Crossed diplopia, Græfe's test with dot and line showed inability to obtain two dots on one line.

V.R.E. cum. + 2.5 cyl. vert. = $\frac{5}{6}$.

V.L.E. cum. + 2.25 cyl. vert. = $\frac{5}{6}$.

F.B., *æt.* 21.—Divergent concomitant squint noticed since birth. Can fix with either eye.

Faint binocular vision obtained.

Convergence fair.

R.E.V. cum. - 6.5 sph. = $\frac{5}{6}$.

L.E.V. cum. - 4.5 sph. - 2 cyl. axis 10° = $\frac{5}{6}$.

Only uses one eye at a time.

A. G., æt. 22.—Divergent concomitant squint. No binocular vision.

V.R.E. = $\angle_{80}^{\frac{1}{8}}$ cum. - 9 = $\frac{1}{8}$.

V.L.E. = $\angle_{80}^{\frac{1}{8}}$ cum. - 13 = $\frac{1}{8}$.

E. F., æt. 10.—Convergent concomitant squint. Can fix with either eye, prefers to fix with left. Alternating squint, more marked on convergence, almost parallelism at distance.

B.E. cum + 3.5 D sph.
+ 3 D cyl. vert. = $\frac{1}{8}$

Cases with amblyopia of one eye and defective binocular vision.

S. C., æt. 13.—Convergent concomitant squint, noticed many years. Fixes with left eye, can fix with the right eye. No binocular vision. No fundus change. Both eyes have five diopters of hypermetropia.

V.R.E. cum. + 5 = $\angle_{80}^{\frac{1}{8}}$.

V.L.E. cum. + 5 = $\frac{1}{8}$.

F. C., æt. 15.—Divergent concomitant squint. No binocular vision. Movements good. Fixes with the right eye. Tenotomy five years ago.

V.R.E. cum. + 1 = $\frac{1}{8}$. V.L.E. fingers at 5 feet.

<div style="display: flex; align-items: center; justify-content: center;"> <div style="margin-right: 10px;">Atropine</div> <div style="border-left: 1px solid black; padding-left: 10px; text-align: center;"> <div style="margin-bottom: 5px;">+1</div> <div style="margin-top: 5px;">+1</div> </div> <div style="margin-left: 10px;">R. E.</div> </div>	<div style="display: flex; align-items: center; justify-content: center;"> <div style="margin-right: 10px;"></div> <div style="border-left: 1px solid black; padding-left: 10px; text-align: center;"> <div style="margin-bottom: 5px;">+1</div> <div style="margin-top: 5px;">+1.5</div> </div> <div style="margin-left: 10px;">L. E.</div> </div>
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Fundi normal.

G. F., æt. 13.—Convergent concomitant squint. Can fix with each eye centrally, but always fixes with the right eye.

Field of vision is defective at outer part of left eye, which is amblyopic.

R.E.V. cum. + 5 = $\frac{1}{8}$. L.E.V. cum. + 5 = fingers at 1 foot.

Atropine

+5
+5.5

Bad binocular vision, only obtained with difficulty.

Cases having gross fundus lesions of one eye, or opacities of the transparent media, with defective binocular vision.

A. L., æt. 8.—Convergent concomitant strabismus. Appearing since inflammation of right eye. Fixes with the left eye.

V.R.E. cum. + 3 = $\frac{1}{8}$ has corneal nebula.

V.L.E. cum. + 3 = $\frac{1}{8}$.

W. P., æt. 7.—Divergent concomitant squint. Fixes with right eye. No binocular vision.

R.E. + .75 = $\frac{1}{8}$. L.E., $\angle_{80}^{\frac{1}{8}}$, not improved.

The fundus of the left eye shows extensive choroidal atrophy.

S.C., æt. 30.—Divergent concomitant squint. The vision of the right eye has been known to be defective since the patient was twelve years old.

R.E. $\angle_{80}^{\frac{1}{8}}$, not improved. Large central patch of choroidal atrophy.

L.E.V. cum. + 1 = $\frac{1}{8}$.

No binocular vision. Movement good. Fixes only with left eye.

F. L., æt. 10. Convergent concomitant strabismus. Fixes with the left eye. Squint more marked during convergence. Parallelism at distance. Noticed since birth. No binocular vision.

By retinoscopy,

$$\begin{array}{ccc} & + 4.5 & \\ & \diagdown & \diagup \\ \text{B.E.} & & + 6.5 \\ & \diagup & \diagdown \end{array}$$

R.E. fingers at 2 feet, not further improved.

$$\begin{array}{l} \text{L.E. cum. } + 4 \\ + 2 \text{ cyl. } 135^\circ = \frac{2}{3} \text{ some.} \end{array}$$

Fundus changes in the right eye. Along course of superior nasal artery numerous patches of choroido-retinitis, looking like remains of old hæmorrhages, disturbing deep parts of retina.

Cases with good vision in each eye. Binocular vision present, and ametropia.

K. D., æt. 40.—Divergent concomitant squint. Fixes with either eye, usually the left. Has binocular vision.

$$\begin{array}{l} \text{R.E.V. cum. } + 1.5 \text{ sph.} \\ + 1.5 \text{ cyl. vert.} \end{array} = \frac{2}{3}$$

$$\begin{array}{l} \text{L.E.V. cum. } + 1.5 \text{ sph.} \\ + 1.5 \text{ cyl. vert.} \end{array} = \frac{2}{3}$$

B. K., æt. 6½.—Convergent concomitant squint. Noticed when two years old.

$$\begin{array}{c} +6 \\ | \\ \hline \\ | \\ +6 \end{array}$$

$$\begin{array}{c} +6 \\ | \\ \hline \\ | \\ +6 \end{array}$$

V. cum. 5 D sph. = $\frac{2}{3}$ each eye.

H. P., æt. 27.—Divergent concomitant squint. Fixes usually with left eye, can fix with right. Has binocular vision for distance and near work.

$$\text{V.R.E. cum. } - 13 = \frac{2}{3}. \quad \text{V.L.F. cum. } - 7 = \frac{1}{3}.$$

Squint noticed since eight years old.

E. M., æt. 18.—Divergent concomitant strabismus. Complains of diplopia and difficulty in reading. Convergence moderate when effort is made. Divergence takes place usually when reading. Fixes with either eye.

$$\begin{array}{c} -3.5 \\ | \\ \hline \\ | \\ -5 \end{array}$$

$$\begin{array}{c} -3.5 \\ | \\ \hline \\ | \\ -2.5 \end{array}$$

$$\begin{array}{l} \text{R.E. cum. } - 4 \text{ D sph.} \\ - 1 \text{ cyl. vert.} \end{array} = \frac{2}{3} \text{ some.} \quad \begin{array}{l} \text{L.E. cum. } - 3 \text{ D sph.} \\ - 1 \text{ horiz.} \end{array} = \frac{2}{3} \text{ some}$$

W. Ross, æt. 34.—Divergent squint noticed six months. V.R.E. fingers at five metres. Left eye shadows at three inches. Fixes with right eye. Optic atrophy, B.E.

M. T., æt. 11.—Paralysis of accommodation. Complains of diplopia. Divergent concomitant squint. Divergence not marked. Crossed diplopia, with Maddox rod.

V.R.E. = $\frac{3}{4}$, cum. + 1 = $\frac{7}{4}$. V.L.E. = $\frac{3}{4}$, cum. + 1 = $\frac{7}{4}$. Reads J 1 cum. + 5 D sph. at six inches.

Pupils react very slightly to convergence. States that he had an illness last March, and dates all his trouble since then. Under a mydriatic he has about 1·5 hypermetropia.

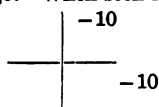
H. S., æt. 14.—Convergent concomitant squint.

R.E.V. = $< \frac{8}{6}$ cum. - 8 D sph. = $\frac{2}{3}$ partly.

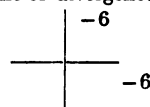
L.E.V. = $\frac{3}{8}$ cum. - 7 D sph. = $\frac{1}{8}$ partly.

Internal rectus of right eye, and subsequently of left eye, divided about two years ago. When seen last the condition was one of divergence.

Atropine



R.E. cum. - 11 = $\frac{1}{4}$.



L.E. cum. - 5·5 = $\frac{1}{2}$.

Binocular vision only at distance. No convergence for near.

P. H., æt. 31.—Divergent and altitudinal squint. States that when he was five years old he remembers being taken to Dr. Walker, of Edinburgh, on account of seeing double, and he believes he has had the trouble all his life. Used to be teased at school about it.

R.E.V. = $\frac{1}{8}$

L.E.V. = $\frac{1}{8}$

R.E. cum. $\frac{-1 \text{ sph.}}{-\cdot 5 \text{ cyl. } 60^\circ} = \frac{1}{8}$

L.E. $\frac{-\cdot 5 \text{ sph.}}{-2\cdot 5 \text{ cyl. axis. } 30^\circ} = \frac{1}{8}$

Patient has no power of fusing one image with the other.

By means of prisms with apex down in front of the left eye about three inches the images are level, and by using prisms horizontal, the images are approximated but cannot be fused and held as one, since they appear to oscillate when they get close together.

This case, then, is one where the visual activity remained during the early period of life equal in the two eyes. Binocular vision normally developed, but fusion of the two images prevented by the presence of an altitudinal squint which he was not able to overcome by any effort.

W. H. N., æt. 31.—Had a convergent concomitant squint when a child, and was operated on, tenotomy being performed. The left eye was always defective, the right eye being good until the accident.

During August, 1900, he received a blow on the right eye from a cork of an exploding soda water bottle, and in consequence of this accident lost the sight of the right eye entirely, having only P. L. Since that time he has been under observation, and as his sight in the left was very indifferent he was obliged to change his occupation.

V.E.E. cum. - 2 cyl. O = $\frac{6}{24}$.

5. N. H., æt. 6.

V.R.E. = $\frac{6}{8}$. Not improved. Emmetropic.

V.L.E. = $< \frac{6}{80}$	$\begin{array}{c} \\ \hline - 5 \\ \\ - 3 \end{array}$	$\begin{array}{r} \text{cum.} - 4 \text{ sph} \\ - 2 \text{ cyl. O} \end{array} = \frac{6}{36}$
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6. O. H., æt. $4\frac{1}{2}$.

E.E. vision = $\frac{6}{80}$ cum. - 3 D sph. = $\frac{6}{24}$.

7. Æt. 3.—Vision, as far as could be discovered, good.

No gross fundus lesion in any of these cases.

SPECIMENS RECENTLY ADDED TO THE PATHOLOGICAL MUSEUM.

By LAURISTON E. SHAW, M.D.,

AND

E. COOPER PERRY, M.D.

PERICARDIUM.

S. 137 Sarcoma of the Pericardium.

A heart divided to shew its ventricles encased in an envelope of soft material, in some places an inch in thickness, which consists of the pericardium infiltrated by a vascular new growth. There is a similar layer around the right auricle, the cavity of which is invaded and its wall almost entirely replaced by the deposit. The glands in the mediastinum are greatly enlarged, and the right bronchus is narrowed. Histologically the growth is a small round-celled sarcoma. The heart with its investment weighs about 30 ounces.

Presented by MR. W. F. CLOWES, 1892.

S. 369 Localised Fibroid Pericarditis.

A portion of a right auricle and ventricle shewing a thickened and wrinkled condition of the epicardium covering the former, whilst on the latter are two fibroid patches somewhat resembling cheloid. Histologically the

fibroid tissue of which the patches consist is seen to infiltrate the myocardium.

From a man who died in a tramcar on his way from an infirmary which he had just left, contrary to the advice of the medical officers. At the autopsy the heart was found to weigh $19\frac{1}{2}$ ounces. The mitral valve was contracted and the myocardium fibroid. There was circumscribed pericardial adhesion over the lower part of the left ventricle. The liver was nutmegged and the kidneys were granular. *See Insp.*, 1894, No. 293.

S. 151 Fibroid Nodules on the Pericardium.

A portion of the pericardium covering the right auricle with the corresponding part of the parietal layer of the sac. The epicardium of the auricle is roughened and rugose, whilst upon the surface which was in apposition to it is seen a cluster of flattened nodules elevated about $\frac{1}{8}$ of an inch above the surrounding surface, and each measuring a line in diameter. Histologically these nodules have a fibrous structure.

Augustus B., æt. 30, was admitted under Dr. Goodhart for hæmoptysis, and died about three months later from phthisis. At the autopsy both pleuræ were found to be firmly adherent, and the lungs were affected with extensive tuberculous disease. *See Insp.*, 1892, No. 272.

S. 489 Acute Pericarditis. Epithelioma of Œsophagus.

A heart with the great vessels and portions of the trachea and Œsophagus. The interior of the pericardial sac is seen to be covered with a thick deposit of recent lymph. At the lower end of the Œsophagus is a deep epitheliomatous ulcer from the base of which two rods have been passed, one into the right bronchus and the other into the pericardial cavity close to the right auricle. The right lung, which is adherent to the Œsophagus, is infiltrated by growth.

Mary Ann M., æt. 60, was admitted under Dr. Shaw for dysphagia, having suffered from severe pain in the chest for four months. She died six days after admission, and at the autopsy patches of broncho-pneumonia were found in the right lung, and there were secondary deposits of growth in the mediastinal glands. *See Insp.*, 1895, No. 178.

S. 627 Pericardium invaded by Sarcoma.

A heart with the great vessels and lower end of the trachea mounted to show the pericardial sac invaded and

partially destroyed by a ragged growth, apparently arising at the lower part of the anterior mediastinum. The epicardium is roughened and shaggy. On the reverse of the specimen the superior vena cava is seen to be occluded by a thrombus which extends into the right auricle. The lungs are adherent to the pleural surface of the pericardial sac. Histologically, the growth is a round-celled sarcoma.

Maria M., æt. 71, was admitted into the Poplar and Stepney Sick Asylum for rheumatism. Three days later œdema of the face and arms suddenly came on, associated with cyanosis. The cardiac dulness was found to be increased and there was a systolic murmur audible over the whole of the præcordial area. The patient died about six weeks from the onset of acute symptoms.

Presented by Mr. CHARLES SPURRELL, 1896.

O.S. 642 Hydro-pericardium.

A pericardial sac, from which the heart has been removed. The sac is uniformly dilated, measuring seven inches from above downwards and five inches transversely. In the recent state it contained a pint and a-half of clear serum. The wall is thin, and presents no evidence of old or recent inflammation.

John R., æt. 39, was admitted under Dr. Goodhart with general dropsy due to aortic incompetence. The cardiac impulse was felt two inches outside the nipple line, and there was considerable increase of præcordial dulness. The patient died one month after admission and at the autopsy the aortic valves were found to be thickened and rigid. The heart weighed 22 ounces. Both pleural cavities contained serum. *See Insp.*, 1889, No. 246.

S. 662 Adherent and Calcareous Pericardium.

A heart divided by a frontal section, and seen from behind to be enveloped in a rough fibrous membrane representing the thickened and adherent pericardium. In the groove between the right auricle and ventricle, and partly embedded in the wall of the latter, is a calcified gumma measuring rather less than half an inch in diameter. In the neighbourhood of the gumma and elsewhere the thickened pericardium contains calcareous plates. The exterior of the sac shows adhesion to the lungs, and there is a mass of fibro-fatty tissue uniting it to the diaphragm. The heart is of normal size; the valves and myocardium are healthy.

Thomas B., æt. 27, was admitted under Dr. Pye-Smith for dyspnœa, cyanosis and œdema. Five years previously he is said to have had rheumatic fever, followed four months later by bronchitis, hæmoptysis and jaundice. Subsequently he was on three occasions tapped for ascites. A month after admission he died from cardiac failure, and at the autopsy the peritoneal cavity was found to contain several pints of fluid, the membrane itself being thickened and the mesentery shortened. The pleuræ were universally adherent, and the tissues of the mediastina were thick and fibrous. *See Insp.*, 1896, No. 471.

S. 717 Chronic Pericarditis.

The base of a heart, seen from behind, and mounted to show at the reflection of the pericardial sac several irregular fibrous tags, the largest measuring an inch in length. The pericardium over the left auricular appendix is thickened. On the reverse of the specimen recent vegetations are seen upon the aortic valves. There is mitral stenosis and the left auricle is hypertrophied.

Hilda F., æt. 23, was admitted under Dr. Hale White with chronic valvular disease of the heart, from which, on the following day, she died. At the autopsy the heart was found to weigh 21 ounces, and there were recent vegetations on the tricuspid and mitral valves. The patient had been in the hospital suffering from acute pericarditis about three years before her death. *See Insp.*, 1897, No. 216.

O.S. 781 Acute Pericarditis.

A heart exposed in its pericardial sac to shew both serous surfaces to be covered by a rugose layer of recent lymph. In the fresh state the sac contained 16 ounces of blood-stained fluid. Around the great vessels and at the bifurcation of the trachea the lymphatic glands are seen to be very greatly enlarged by a deposit which histologically consists of small round cells and numerous giant cells with a few foci of caseation.

Mary Ann T., æt. 34, was admitted under Mr. Jacobson for a swelling on the right side of her neck, which had been slowly increasing in size for the preceding nine months. On admission, she was found to be suffering from pericardial effusion, for the relief of which her chest was twice aspirated. She died a week after admission, and at the autopsy the cervical and mediastinal glands were found to be enlarged, and there was considerable serous effusion into the pleural and peritoneal cavities. *See Insp.*, 1890, No. 101.

O.S. 859 Sarcoma of the Pericardium.

A heart shewing beneath the epicardium on the front of the right ventricle a flattened nodule measuring a third of an inch in diameter.

Robert R., æt. 54, was admitted under Dr. Hale White for malignant growth of the liver secondary to a sarcomatous tumour which had been excised from the right arm six months previously. He died one month after admission, and at the autopsy secondary deposits were found in the lungs, liver, spleen and peritoneum. *See Insp.*, 1890, No. 329; and *Prep.* 1525.

O.S. 914 Adherent Pericardium. Chronic Endocarditis.

A heart the surface of which shews numerous flocculent tags left after the partial removal of the adherent parietal pericardium, a portion of which is still seen attached over the right auricle. The aortic, mitral and tricuspid valves are fibrous and stenosed.

Alice L., æt. 18, was admitted under Dr. Shaw for ascites and dyspnoea due to heart disease. She had had numerous attacks of chorea since the age of four, and one attack of acute rheumatism. She died about four months after admission, and at the autopsy the heart was found to weigh 16 ounces, and the lungs were splenised. The liver was cirrhotic, the spleen enlarged, and the kidneys were indurated. *See Insp.*, 1890, No. 474.

HEART.

S. 2 Perforation of Mitral Valve. Ulcerative Endocarditis

A portion of a heart mounted to shew a small perforation close to the free border of the anterior flap of the mitral valve. Hanging immediately above it is a pendulous mass of fibrinous deposit attached to one of the aortic cusps. Similar smaller masses adhere to the other cusps, which are in parts destroyed by ulceration, and elsewhere thickened by chronic endocarditis. On the reverse of the specimen the perforation is seen to be surrounded by a raised ring of vegetations.

John P., æt. 29, was admitted under Dr. Hale White with fever and the physical signs of disease of the lungs and heart. He had had acute rheumatism six months previously, and had recently suffered from hæmoptysis. He died eight days after admission, and at the autopsy the heart was found to weigh 19 ounces, and both sides were hypertrophied and dilated. There was a cavity at the apex of

the left lung, and tubercle bacilli were found in the caseous material lining its wall. The spleen weighed 15 ounces, and contained a large infarct. *See Insp.* 1891, No. 232.

S. 5 Calcareous Vegetations on the Mitral Valve.

A mitral valve, thickened but not contracted, and mounted to shew upon the auricular aspect of its posterior curtain a cluster of fibro-calcareous vegetations, measuring an inch and a half in length, and projecting half an inch into the auriculo-ventricular orifice. The mass has a cauliflower-like surface and is free from recent deposit.

Jane P., æt. 38, was admitted under Dr. Pitt a week after her confinement, for cardiac palpitation and dyspnoea. There was a systolic murmur at the apex of the heart, associated with a diastolic thrill. The patient died suddenly four days after admission, and at the autopsy the heart was found to weigh 23 ounces, and the left pleural cavity contained about a pint of serum. The right lung contained a large infarct, and the liver was nutmegged. *See Insp.* 1891, No. 256.

S. 97 Incomplete Septum Ventriculorum.

A heart mounted to shew a small opening in the septum ventriculorum, immediately below the right semilunar valve. This valve is considerably thickened, and at the bottom of the pouch which it forms with the aorta there is a second opening close to the first, and, like it, communicating with the right ventricle. As seen from the right ventricle, the second opening is situated in the centre of a pouched membranous diaphragm, and corresponding with the point on which the reflux blood-stream would impinge is a fibrous thickening of the endocardium of the ventricle.

John L., æt. 15, was admitted under Dr. Hale White with symptoms of chronic cardiac failure of about four months' duration, which proved fatal fourteen days after admission. At the autopsy the heart was found to weigh seven and a half ounces, and the kidneys and liver were congested. The lungs contained "apoplexies," and there were patches of recent lymph upon the pleura. *See Insp.* 1892, No. 112; and *Trans. Path. Soc.*, 1892, p. 34.

S. 186 Rupture of the Left Ventricle.

A heart shewing upon its anterior surface close to the septum ventriculorum, an oblique rent three quarters of an

inch in length which communicates with the left ventricle. Immediately above it a smaller superficial rent is seen. There is a considerable excess of fat upon the surface of the organ. The coronary arteries are slightly atheromatous.

S. 194 Fibroid Endocardium.

A portion of a right ventricle shewing over many of the columnæ carneæ a white fibrous thickening of the endocardium. The fibrous material is seen on section not to penetrate the deeper layers of the muscle.

Joseph D., æt. 42, was admitted under Dr. Hale White for hæmoptysis, due to phthisis, from symptoms of which he had suffered for seven years. Ascites and œdema of the lower extremities had existed for six months. The abdomen was tapped twenty-seven times before his death, which occurred eleven months after his admission to the hospital. Whilst under observation a systolic bruit developed, which was best heard between the apex of the heart and the sternum. At the autopsy, both lungs were found to be affected with fibroid phthisis; there was general chronic peritonitis, and the liver was nutmegged. The heart weighed 19 ounces, and the left ventricle was of normal size. The pulmonary artery was atheromatous. See *Insp.*, 1893, No. 16; and *Trans. Path. Soc.*, 1893, p. 24.

S. 197 Polypus in the Left Auricle.

A left auricle laid open to shew upon the inter-auricular septum, above the situation of the fossa ovalis, a smooth rounded polypus measuring a quarter of an inch in diameter. Histologically it consists of a hyaline matrix containing blood-vessels and much brown pigment. On the reverse of the specimen ante-mortem clot is seen in the right auricular appendix.

Mary L., æt. 68, an enormously fat woman, was admitted under Mr. Symonds with a strangulated umbilical hernia, for the relief of which herniotomy was performed. The patient died twenty-four hours later. See *Insp.*, 1893, No. 18.

S. 262 Partial Laceration of the Left Ventricle.

A heart divided, to shew in the interior of the left ventricle towards the apex, a laceration of the muscle which extends to, but does not perforate the epicardium.

Richard E., æt. 48, was admitted, under Mr. Howse, for injuries caused by some deal planks falling on him. He died about twenty-four hours after the accident, and at the autopsy the sternum and three of

the ribs on the left side were found to be fractured. Blood was extravasated into the tissue of the parietal pericardium and the anterior mediastinum. There was a large ecchymosis on the surface of the heart, corresponding to the laceration. There was no blood in the pericardial sac. *See Insp.*, 1893, No. 265.

S. 293 Atheromatous Coronary Arteries.

A heart with the first part of the aorta laid open to shew the interior of the vessel extensively affected by atheroma. The orifices of the coronary arteries are narrowed, the left being almost entirely occluded. On the reverse of the specimen, the arteries which have been laid open are seen to be atheromatous, but in a much less degree than is the aorta. The aortic valves are somewhat thickened but appear to have been competent. Histological examination of the myocardium shows that many of the fibres have undergone fatty and hyaline degeneration.

James C., æt. 59, was admitted under Dr. Washbourn for dyspnoea, for the relief of which paracentesis thoracis was performed. He had been attending as an out-patient for many months, suffering from angina pectoris. At the autopsy the heart, which was below the average weight, showed slight dilatation of the left ventricle. The arch of the aorta was dilated. The orifice of the cœliac axis was obliterated by calcareous deposit. There was chronic interstitial nephritis, the renal arteries being atheromatous. *See Insp.*, 1894. No. 4.

S. 328 Punctured Wound of the Right Ventricle.

A heart shewing upon its anterior surface about the middle of the right ventricle a linear wound measuring three-eighths of an inch in length, which penetrates the wall. Corresponding in position there is a small superficial cut in the interventricular septum.

From a boy, æt. 11, who, while playing with a pocket knife, stabbed himself in the præcordial region, and died in three minutes.

Presented by MR. KNYVETT, 1894.

O.S. 358 Suppurative Myocarditis.

The heart of a child mounted to shew the cut surface of the muscle of the left ventricle to have a somewhat mottled appearance, the muscle in parts being replaced by a soft white material. The valves and epicardium are normal.

Histologically the myocardium is seen to be in a condition of diffuse purulent infiltration.

From a child *æt.* 3½ years who died in the Evelina Hospital with symptoms of nephritis one month after the onset of an attack of scarlet fever. At the autopsy the cavity of the left ventricle was found to be somewhat dilated, and its wall very much thickened by a diffuse infiltration of pus. There was no lymph on the surface of the valves or pericardium. The kidneys were affected by tubal nephritis. *See Trans. Path. Soc.*, 1880, p. 70.

Presented by DR. GOODHART.

S. 366 Hypertrophy and Dilatation of the Heart.

A heart with its cavities laid open to show the left ventricle enormously dilated, whilst its wall is of about the normal thickness. In the recent state the heart weighed 32 ounces, and the hypertrophy and dilatation affected both sides equally. On the reverse of the specimen the epicardium is seen to be free from adhesion, opaque and thickened. Histological examination shews that the muscle of the left ventricle is normal.

Robert L., a painter, was admitted under Dr. Taylor for dyspnoea and attacks of faintness, from which he had suffered at intervals for two years. There was a history of gout and lead poisoning, but none of syphilis or rheumatism. On admission, there were physical signs of great enlargement of the heart, without bruit or albuminuria. Three days after admission the patient died, and at the autopsy 50 ounces of serous fluid were found in the right pleural cavity and 30 ounces in the pericardial sac. The valves of the heart were normal and the aorta was not atheromatous. The kidneys were healthy. *See Insp.*, 1894, No. 272.

S. 447 Mitral Stenosis. Thrombus in Auricular Appendix.

A section through a heart shewing the mitral valve to be funnel-shaped, and so narrowed as barely to admit the tip of the little finger. The left auricle is dilated and slightly thickened, and its appendix is distended with ante-mortem thrombus.

Edith P., *æt.* 26, was admitted under Dr. Galabin in order that labour might be induced for the relief of the symptoms of chronic cardiac failure, which had gradually been increasing during her pregnancy. She had suffered from chorea every year from the age of 7 to 14. Labour was induced three days after admission, and the patient died four days later. At the autopsy the heart was found to weigh 17 ounces, both pleural cavities contained serum, and there were several recent infarcts in the base of the right lung. The kidneys were scarred. *See Insp.*, 1895, No. 127.

E. 464 Ulcerative Endocarditis. Aneurysms of the Heart.

A portion of a heart mounted to shew extensive fibro-calcareous changes in the aortic valve. There is a large laceration in the free edge of the right anterior cusp, surrounded by vegetations. There are small aneurysms between the two anterior cusps and between the posterior and left anterior cusp.

Nathaniel F., *æt.* 59, was admitted under Dr. Goodhart with apoplectic symptoms, and died in a comatose condition fourteen hours after admission. At the autopsy there was an aneurysm of the right middle cerebral artery at its main bifurcation, together with a large hæmorrhage in the substance of the brain. The heart weighed 16½ ounces. *See Insp.* 1894, No. 469.

O.S. 472 Aneurysm of the Heart.

A heart divided to shew the left ventricle converted into an aneurysmal cavity, which measures five inches in its longest diameter, and is almost filled with adherent thrombus. The muscle of the interventricular septum is of normal thickness, but elsewhere the myocardium is greatly thinned, and at the apex of the heart is entirely replaced by fibrous tissue. The epicardium, which is very thick, is everywhere roughened by old adhesions.

From Thomas O., *æt.* 42, who was found dead in the streets. He was said to have had syphilis, and to have been an in-patient in the London Hospital two or three years before his death. At the autopsy the heart was found to weigh 18 ounces; there was a scar upon the glans penis, and one of the testes was fibroid. *See Insp.* 1888, No. 287.

O.S. 479 Mitral Stenosis. Thrombus in the Auricle.

The base of a heart with the left auricle laid open to shew a ragged mass of clot, almost entirely filling the cavity and continuous with firmer thrombus distending the appendix. The mitral orifice is contracted so as barely to admit two fingers.

Sarah P., *æt.* 50, was admitted under Dr. Pavy for palpitation of the heart and dyspnoea, having three weeks previously suffered from hæmatemesis. On admission, the heart's action was noticed to be irregular, and a presystolic bruit was heard at the position of the cardiac impulse. The patient died three weeks later, and at the autopsy the heart was found to weigh 14 ounces and the kidneys were scarred. *See Insp.* 1888, No. 307.

S.500 Dilatation of Left Ventricle.

A child's heart mounted to shew acute dilatation of the left ventricle. The valves and pericardium are normal. Histological examination shews the muscle of the left ventricle to be healthy. In the recent state the heart weighed three ounces.

William J., æt. 4, was admitted under Dr. Shaw for tetanus, following a punctured wound of a toe, and died six days after the onset of symptoms. *See Insp.* 1895, No. 196.

S. 532 Hypertrophy of the Heart. Myocarditis.

A heart, which in the recent state weighed 18 ounces, mounted to shew considerable hypertrophy of its walls with some enlargement of the right side. The valves are healthy and the aorta is free from atheroma. Histological examination of the left ventricle shews that the muscle fibres are swollen, have lost their striation, and are separated from each other by small-celled infiltration.

Thomas S., æt. 49, was admitted under Mr. Howse with pyrexia and œdema of the lungs. A fortnight previously he had been in the hospital for a few days with a contused wound of the scalp and symptoms of concussion. After leaving the hospital he had been drinking heavily, and on the day following his re-admission he became delirious. Two days later, whilst straining at stool, he fell back and died instantly. At the autopsy patches of softening were found in the cerebral cortex, the liver was nutmegged, and the kidneys, which weighed 16 ounces, were in a condition of cyanotic induration. *See Insp.*, 1895, No. 350.

S. 551 Aneurysms of the Heart.

A portion of a heart mounted to shew two sacculated aneurysms, the orifices of which are situated just below the two extremities of the posterior aortic cusp. The one on the right admits the tip of the little finger, and the overhanging right semilunar cusp has a wide perforation, through which the regurgitant blood-stream apparently impinged upon the wall of the sac. On the left side an oval opening three-quarters of an inch in diameter leads into a sac about as large as a walnut, which bulges into the left auricle. The aortic valves are calcareous, distorted, and perforated in several places. The mitral valve is somewhat thickened, and there are fibroid patches in the myocardium.

From the Dissecting Room, 1889.

O.S. 566 Atrophy of the Heart.

A heart, which in the recent state weighed four and a-half ounces, mounted to shew the atrophy of the organ associated with chronic wasting diseases. There is a moderate deposit of fat on the surface of the right ventricle.

William S., *æt.* 24, was admitted under Mr. Durham for a psoas abscess, having suffered for five years from symptoms of spinal caries. He died about three months after admission, and at the autopsy the liver, kidneys, spleen and intestines were found to be affected with lardaceous disease. The liver, which weighed 68 ounces, was very fatty. *See Insp.*, 1889, No. 28.

S. 600 Aneurysm of the Mitral Valve.

The left ventricle of a heart, laid open to shew at the upper part of the aortic cusp of the mitral valve a pouch, which admits the tip of the middle finger and projects somewhat into the auricle. A little above the aneurysm is seen a second slightly larger sac, caused by a protrusion of one of the sinuses of valsalva. The aortic valves are thickened, and were incompetent. The ventricle is dilated.

William C., *æt.* 44, was admitted under Dr. Pye-Smith, for cardiac pain and palpitation, from which he had suffered at intervals for three years. On admission physical signs of enlargement of the heart were detected, associated with a loud to-and-fro aortic bruit. The patient became delirious, and died ten days after admission. At the autopsy, the heart was found to weigh 29½ ounces and many of the arteries were atheromatous. *See Insp.*, 1896, No. 66.

O.S. 624 Lacerations of the Left Ventricle.

A heart, in the dilated left ventricle of which are seen two ragged lacerations extending about a third of an inch into the cardiac muscle. One of them is situated at the lowest part of the interventricular septum, and the other, which is somewhat higher, crosses the base of the papillary muscle arising from the anterior wall. On the exterior of the heart, corresponding closely to this latter laceration, is a rent with gaping edges about an inch in length, in the upper margin of which is seen the torn end of a coronary vein.

Jacob N., *æt.* 35, was admitted under Mr. Howse, having been struck by the pole of a van on the left side of the chest. About five hours later he died, and at the autopsy several ribs were found to be fractured, and there were about 14 ounces of unclotted blood in the pericardium. *See Insp.*, 1889, No. 202.

O.S. 654 Sarcoma of the Heart.

A portion of the right side of a heart mounted to shew an oval mass of growth about an inch in length, situated beneath the epicardium at the base of the right ventricle. The growth, which projects as a convex nodule from the surface of the heart, infiltrates the superficial layers of the cardiac muscle. It has the histological structure of a round and oval-celled sarcoma.

Michael S., æt. 60, was admitted under Dr. Pitt with the physical signs of a malignant growth of the mediastinum and effusion into the left pleural cavity. He died a fortnight after admission, and at the autopsy sarcomatous deposits were found in the thoracic glands and in the liver and kidneys. *See Insp.*, 1889, No. 288.

O.S. 689 Atrophy of the Heart.

A heart weighing six ounces and mounted to shew the condition of general atrophy. The epicardium is wrinkled, and the coronary arteries are prominent. In the recent state there was œdema of the epicardial connective tissue with an almost total absence of fat.

Joseph T., æt. 55, was admitted under Dr. Perry with symptoms of cancer of the stomach, having suffered from gastric symptoms for three years. Œdema of the extremities supervened, and the patient died eleven weeks after admission. At the autopsy the body was emaciated, and the liver, which weighed only 28 ounces, contained numerous small secondary deposits. *See Insp.*, 1889, No. 387.

S. 703 Pyæmic Abscesses of the Heart. Perforation of Mitral Valve.

A portion of a heart mounted to shew two small round patches of sessile vegetations upon the ventricular surface of the posterior flap of the mitral valve. In the middle of the larger patch is seen a perforation through which a blue rod has been passed, traversing the valve and entering a small abscess cavity in the wall of the ventricle immediately beneath the aortic orifice. The cut surface of the ventricle presents several other small points of suppuration.

Thomas R., æt. 13, was admitted under Dr. Pye-Smith with symptoms of pyæmia, and died thirty-six hours after admission, on the ninth day of his illness. At the autopsy a collection of pus was found separating the periosteum from the neck of the right femur, and the liver and kidneys contained numerous small abscesses. *See Insp.*, 1897, No. 106.

S. 715 Gumma invading the Right Ventricle.

A heart laid open to shew in the wall of the right ventricle, immediately below the semilunar valves, a large mass of gummatous material projecting into the cavity of the ventricle, and partially obstructing the pulmonary artery. The deposit is continuous with a mass of fibro-caseous material which surrounds and compresses the great vessels at the base of the heart. The epicardium of the right side of the heart is roughened, and was adherent.

From Hubert H., æt. circa 30, who died quite suddenly. A few weeks before his death he underwent an operation for piles, and was then thought to be in good general health. At the autopsy all the viscera with the exception of the heart were found to be normal. *See Insp.*, 1897, No. 206.

O.S. 844 Thrombus in the Ventricles.

A heart with its ventricles laid open to shew in each of them a considerable deposition of thrombus amongst the meshes of the columnæ carneæ. In the left ventricle the thrombus presents numerous polypoid excrescences, some of which shew central excavation. On the right side, where the clot is less abundant, a cylindrical mass is seen adherent to the apex of the ventricle. The heart weighs 16 ounces. The valves are normal, and there is no clot in the auricles.

Jane H., æt. 27, was admitted under Dr. Shaw for œdema of the legs and of the left arm. She had been confined six week previously, and on admission the præcordial dulness was found to be increased to the right of the sternum. Six days later she died, and at the autopsy a large infarct was found in the anterior edge of the right lung, with emboli in the spleen and kidneys. There was thrombosis of the axillary, brachial and jugular veins on the left side. *See Insp.*, 1890, No. 293.

O.S. 871 Endocarditis of the Aortic Valves.

A portion of a left ventricle laid open to shew the aortic valve affected by chronic and acute endocarditis. The cusps are thickened and their free borders are rounded, that of the posterior cusp being capped by a row of minute vegetations. Similar vegetations are seen along the line of contact of the other cusps,

Thomas H., æt. 22, was admitted under Dr. Shaw on the fifth day of an attack of acute rheumatism, with a systolic bruit at the apex of the heart. The patient subsequently developed physical signs of pleurisy, pericarditis and double pneumonia, and died nineteen days after admission. At the autopsy the heart was found to weigh 17 ounces, and the pericardial sac was obliterated by recent adhesions. There were vegetations on the mitral valve. *See Insp.*, 1890, No. 367.

O.S. 954 Aneurysm of the Heart.

A heart, at the apex of which is seen a thin-walled globular sac, measuring an inch and a half in diameter, and communicating with the left ventricle by a narrow opening. The walls of the sac are formed of fibrous tissue, to which the parietal pericardium is, in part, adherent. The endocardium of the ventricle is much thickened. Histological examination of the muscle in the neighbourhood of the apex shews fibroid change. The aorta is roughened by atheroma.

Richard W., æt. 28, was brought in dead. He was said to have been under treatment for syphilis. While following his occupation as a labourer he attempted to lift a heavy load, and immediately expired. At the autopsy, the heart was found to weigh 16 ounces, and the pericardial sac was distended with 21 ounces of fluid and clotted blood, which had escaped from a small V-shaped laceration of the aneurysm. Both testes were fibroid. *See Insp.*, 1891, No. 72; and *Trans. Path. Soc.*, 1891, p. 61.

O.S. 981 Thrombosis of the Left Auricle.

A section through the left auricle and ventricle, shewing the cavity of the auricle to be almost filled with a laminated and convoluted thrombus which is adherent to the wall at its upper part. Smaller masses of clot are seen in the appendix, and between the thrombus above mentioned and the mitral valve. The valve is fibrous and greatly contracted. The wall of the auricle is hypertrophied.

Robert C., æt. 45, was admitted under Dr. Perry with a presystolic bruit and signs of fluid in the right pleural cavity. Eight days later he died, and at the autopsy an infarct was found in the right lung, and the viscera were congested. The heart weighed 14 ounces, and the tricuspid valve was somewhat contracted. There were small thrombi in the right auricular appendix. *See Insp.*, 1891, No. 149.

O.S. 982 Aortic and Mitral Incompetence.

A portion of a heart, laid open to shew the aortic valve affected by chronic inflammation. The cusps are thickened,

rigid and deformed; they are partially coherent to each other, and between the posterior and left cusps is seen a row of minute vegetations. The mitral valve is greatly thickened, and was incompetent.

John O., *æt.* 37, was admitted under Dr. Goodhart, with signs of chronic cardiac failure. He had had rheumatic fever eight years previously. On admission, a systolic bruit was heard at the apex of the heart, and there was an inconstant diastolic bruit at the base. The patient died from lobar pneumonia about six weeks after his admission. At the autopsy the heart was found to be hypertrophied and dilated, weighing 24 ounces. The pericardium was adherent. The liver and kidneys were congested. *See Insp.*, 1891, No. 139.

O.S. 984 Endocarditis in Lobar Pneumonia.

An aortic valve mounted to shew upon the corpus arantii of the right cusp a prominent vegetation about the size of a swan shot. The cusps are otherwise normal.

Joseph R., *æt.* 50, was admitted under Dr. Perry on the fifth day after the onset of symptoms of acute pneumonia with the physical signs of consolidation of the right apex. A fortnight after admission he became delirious, and a week later he died. At the autopsy the upper part of the right lung was found in a condition of acute interstitial pneumonia, and there was early suppurative meningitis. Pneumococci were found in the lung, meninges, and in the aortic vegetations. *See Insp.*, 1891, No. 164.

S. 98/46 Atheroma of Aorta and Aortic Valves.

A portion of a left ventricle with the first part of the aorta mounted to shew the vessel and the cusps of the aortic valve affected by atheroma. In the aorta the disease is limited to the first inch and a half of the vessel, the wall being thickened and presenting a rough calcareous surface. The cusps are deformed, stretched and thickened, and one of them is partially torn from its attachment.

Henry R., *æt.* 38, was admitted under Dr. Pye-Smith with signs of heart disease. Nine weeks before admission, whilst at work, he was suddenly seized with dyspnoea and symptoms of cardiac failure. On admission, there were physical signs of aortic regurgitation, and of hypertrophy and dilatation of the heart. The left pleural cavity was thrice aspirated. The patient died three weeks after admission, and at the autopsy the heart was found to weigh 24 ounces. The lungs were compressed, the liver nutmegged, and the kidneys indurated. *See Insp.*, 1898, No. 251.

S. 98/47 Melanotic Sarcoma of the Heart.

A heart divided to shew in the anterior wall of the left ventricle a wedge-shaped mass of black growth, the base being towards the epicardium and measuring three quarters of an inch in diameter. There is a small similar deposit beneath the endocardium of the right auricle. Histologically the growth is a sarcoma the cells of which are round and oval and contain much pigment.

Eliza W., æt. 54, was admitted under Dr. Goodhart with symptoms of cerebral tumour. Five years previously Mr. Davies-Colley had removed a growth from the lower jaw which recurred two years after operation. The patient died seven days after her admission, and at the autopsy deposits of melanotic growth were found in the brain, lungs, jejunum and in the mesenteric glands. *See Insp.*, 1898, No. 262.

S. 99/30 Mitral Stenosis. Dilatation of the Auricles.

A heart mounted to show great dilatation of the auricles resulting from mitral stenosis and tricuspid regurgitation. The left auricle measures six inches in its longest diameter, and its walls are slightly thinner than normal. The mitral valve is fibrous, and barely admits the tip of the index finger. On the reverse of the specimen the right auricle is laid open, and measures four inches in its longest diameter. The tricuspid orifice admits four fingers, and the valve has a thickened and rolled edge. The aortic and pulmonary valves are healthy. The right ventricle is hypertrophied and dilated, the left being somewhat below the average size.

Mary R., æt. 47, was admitted under Dr. Pitt with signs of heart disease, having occasionally suffered from dyspnoea and oedema of the ankles for fifteen years. Systolic and diastolic murmurs were heard at the apex and a systolic murmur in the aortic area. The patient died eight days after admission and at the autopsy the heart was found to weigh 16 ounces and the kidneys were scarred. *See Insp.*, 1899, No. 83.

S. 99/87 Pouching of the Inter-auricular Septum. Patent Foramen Ovale.

A heart which weighed 24 ounces, and presents general hypertrophy and dilatation without valvular lesion. In the right auricle is seen a finger-like membranous pouch, measuring about two inches in length and an inch in width,

and having at its apex a circular opening a third of an inch in diameter. An inspection of the left auricle shows that the pouch is formed by a protrusion of the portion of the inter-auricular septum, forming the fossa ovalis.

From a woman, æt. 55, who presented the physical signs of enlargement of the heart, and died from chronic cardiac failure. At the autopsy, the right kidney, which was granular, was found to weigh $7\frac{1}{2}$ ounces, the left being represented by a small mass of fibrous tissue containing a few cysts. The liver was nutmegged, and there was effusion into the pleural and peritoneal cavities.

Presented by Dr. CLAUDE TAYLOR, 1899.

LIST OF SPECIMENS ADDED TO THE DENTAL MUSEUM.

BY J. LEWIN PAYNE.

DENTAL SURGERY SECTION.

I. IRREGULARITIES OF THE TEETH:—

- A. Number.
- B. Size.
- C. Form or structure.
- D. Position.

A. NUMBER.—Thirty-two teeth being the normal number for man any excess or diminution constitutes an irregularity; both of these forms are fairly common, but of the two one more frequently meets with the excess in number.

Excess in number.—Any teeth in excess of the normal may be spoken of as *supernumerary*; but when they differ in no respect from the members of the series in which they are situated they are called *supplemental* teeth.

Supplemental.—For example, increased number of lateral incisors, either upper or lower; excessive number of bicuspid; cases have also been recorded of supplemental molar teeth.

1. Model of the maxillæ taken at the age of $2\frac{1}{2}$ years, showing a supplemental right temporary lateral incisor.

Presented by Mr. TRAER HARRIS.

2. A similar condition seen in a model taken at the age of $4\frac{1}{2}$ years.

3. Model of the upper jaw of a child, aged 7 years, in which three right temporary incisors are seen ; on the left side the temporary incisors are gone, but it was stated that the teeth were quite normal.

Presented by Mr. W. H. MORRIS.

4. Model of a mandible of a girl, aged 8 years, presenting five permanent incisors.

Presented by Mr. A. V. BRIMMER.

5. A supplemental tooth—one of five lower incisors ; three-quarters of the root was exposed on the labial side and its position was apparently that of a lower lateral incisor on the right side.

Presented by Mr. E. C. CLAYTON.

6. Model of the upper jaw, showing a supplemental left upper lateral (chipped).

Presented by Mr. F. NEWLAND-PEDLEY.

7. Models of a mouth showing a supplemental lateral behind the left central incisor, which it has rotated and thrust forward.

Presented by Mr. W. A. MAGGS.

8. Model showing a supplemental upper lateral incisor on the right side.

Presented by Mr. E. HUTSON.

9. A supplemental left upper lateral incisor.

10. Model of the maxillæ with supplemental incisors situated behind the centrals, which are slightly displaced.

Presented by Mr. J. L. PAYNE.

11. Models of a mouth showing a small supplemental bicuspid on the left side of the lower jaw.

Presented by Mr. W. A. MAGGS.

12. Model of a mandible with three bicuspid on each side of the jaw.

Presented by Mr. R. UMNEY.

13. Model showing five lower bicuspid teeth on the left side (Duplicate of one in the Museum of the Victoria Dental Hospital, Manchester).

Presented by Mr. D. HEADRIDGE.

14. A remarkable model of the maxillæ with six bicuspid on the left side, a pyramidal shaped supernumerary tooth behind the central incisors, and another supernumerary gemminated to the right lateral.

15. Two models of maxillæ each having a supernumerary molar situated on the buccal side.

Irregular supernumerary teeth.—These are more common.

Characteristics.—(i.) The lingual and labial surfaces have not necessarily any difference in form. (ii.) The enamel is said to terminate in an even line around the neck of the tooth (generally this is so; but it is not always true). (iii.) Roots are single (with very rare exceptions).

Types.—Conical, pyramidal, and molariform.

16. Model of the upper jaw showing a deciduous supernumerary on the right premaxilla. Miss Y., aged 8.

17. Eight conical supernumerary teeth, some being remarkably small and very imperfectly formed, found in the front of the mouths of different individuals.

Presented by the late Mr. JOSEPH FOX.

18. A progressive series of ten supernumerary teeth commencing with the simple conical type and becoming more complete as the series continues, the last showing an attempt at the formation of cusps. (The root of the third shows signs of absorption.)

Presented by Mr. F. NEWLAND-PEDLEY.

19. Three conical supernumerary teeth.

20. Model of upper jaw showing two conical supernumerary teeth behind the right central incisor, causing displacement of the central and canine.

Presented by Mr. F. NEWLAND-PEDLEY.

21. A series of four models (α , β , γ , δ), of maxillæ, showing conical supernumerary teeth in the incisor region, in most cases displacing central incisors.

22. Model showing a supernumerary in the place of the upper left central incisor, conical in form.

Presented by Mr. G. O. RICHARDS.

23. Conical-shaped supernumerary projecting anteriorly between the upper central incisors.

Presented by Mr. M. F. HOPSON.

24. Three models (α , β , γ) showing conical supernumerary teeth in the median line; α , the dental arch is contracted giving the appearance of a high vault. γ . The cutting edge of the tooth is grooved by attrition of the lower incisors.

25. A model of 2 conical supernumerary teeth situated behind the right and left centrals respectively.

Presented by Mr. F. BLEWITT.

26. A similar model in which the upper centrals and laterals have been displaced.

Presented by Mr. F. E. LAMBERT.

27. Another model in which a supernumerary tooth has displaced a right upper lateral.

Presented by Mr. A. J. COLLETT.

28. A conical supernumerary tooth in the median line, causing considerable irregularity and forming a sort of central point around which the upper central and lateral incisors are arranged.

Presented by Mr. S. R. APHORPE, 1892.

29. Model of a lower jaw with a supernumerary tooth situated in front of the left central incisor.

Presented by Mr. W. R. BUTLER.

30. Three supernumerary teeth in the palate, two being conical and one having three cusps.

Presented by Mr. G. O. RICHARDS.

31. Three supernumerary teeth, one with a cruciform crown and the others having a triangular masticating surface.

Presented by Mr. F. TA'BOIS.

32. Three molariform supernumerary teeth from the back part of the jaws.

33. Model showing two supernumerary teeth of the bicuspid type, in the median line, which are separating the upper central incisors. The crowding has brought about caries in the left canine.

Presented by Mr. F. V. MACKENZIE.

34. Two models of maxillæ, showing cubical-shaped supernumerary teeth, situated behind the incisors.

Presented by Mr. T. S. DAVIDSON.

35. Cubical-shaped supernumerary tooth in median line, causing displacement backwards of the right lateral.

36. Two models, showing large, cubical-shaped supernumerary teeth in the posterior incisive region; in one case the left upper central is displaced forwards.

37. Model of two supernumerary teeth of cubical form, occupying the position of the upper central incisors, the left central seems about to erupt; the right central did erupt, but was so loose that the patient removed it. Patient aged 19 years.

Presented by Mr. A. V. BRIMNER.

38. Model of the maxillæ, with a supernumerary tooth in the region of the third molar.

Presented by Mr. K. GOADBY.

Deficiency.—Deficiency in the number of the teeth may be due to one of two causes:—

(α) Tooth quite unformed.

(β) Tooth, though developed, remaining unerupted.

While in very rare cases there is a total congenital absence of teeth, one not infrequently meets with patients in whom a large number are missing.

Deficiency is more commonly observed in the permanent than in the temporary series, the teeth most often absent being the upper laterals, the third molars, and the second lower bicuspids. Diminution in number resulting from extraction is not, of course, included under this heading.

39. Model showing the absence of a left upper lateral incisor, associated with the presence of a diminutive lateral on the right side.

Presented by Dr. J. W. PARE.

40. A model showing a similar condition.

Presented by Mr. J. L. PAYNE.

41. Model with both upper laterals absent, and having the canine teeth in close proximity to the central incisors on each side. The persistent temporary canines are situated immediately behind the permanent ones.

Presented by Mr. A. E. BAKER.

42. Model of the maxillæ with both lateral incisors missing, and the temporary canine persisting behind its permanent successor on the right side. Patient aged 25 years.

Presented by Mr. E. MORGAN.

43. A similar model, in which the persistent temporary canine is situated in front of its successor on the left side. Patient aged 24 years.

44. The model of the maxillæ of a girl, aged 14 years, showing absence of the first and second left incisors, and having the left temporary canine retained with its permanent successor in front of it. No history of injury or extraction.

Presented by Mr. J. M. C. JACOBS.

45. The left half of an adult mandible, found in the grounds of Guy's Hospital during building operations, which shows the second temporary molar persistent and moderately well implanted, whilst a section of the bone beneath it demonstrates the absence of any sign of a successional tooth.

Presented by Mr. G. S. H. BARNETT.

46. A case of non-eruption of a left upper central incisor in a boy aged 12 years, apparently due to the persistence of a temporary incisor root.

Presented by Mr. C. J. HINCHLIFF.

47. Singular models of upper and lower jaws taken from the mouth of a patient aged 29 years, and showing total absence of bicuspid teeth; the patient stating that she was quite certain none had at any time been erupted or extracted. In the maxillæ, both the second temporary molars are standing, while an interspace exists on the right side between the canine and lateral; in the mandible, wide spaces are seen between the canines and molars and also between the canines and laterals on each side.

Presented by Mr. J. L. PAYNE.

48. Models from the mouth of a boy aged 13 years, the third out of five children, there being nothing unusual in the dentition of the other members of the family and no signs of rachitis or syphilis in him. The father stated positively that no teeth had been extracted. All the molars (except one lower temporary), the second bicuspid, and the left lower central incisor are absent, and the lower right central incisor is quite diminutive.

Presented by Mr. W. A. MAGGS.

49. Models of the upper and lower jaws, shewing unusual deficiency; in the mandible there are only four cone-shaped teeth on each side in front of the mouth and two roots posteriorly; in the maxillæ, there are but five teeth on either side, and a space exists on the left side of the median line.

Presented by Mr. J. H. BADCOCK.

50. Models of the maxillæ and mandible having only four teeth below and six teeth above; of the four lower teeth, two have cone-shaped crowns, one is irregular, and the fourth molariform: of the six upper teeth five are conical.

Presented by Mr. G. H. MORRIS.

51. Models of the maxillæ and mandible presenting only six teeth above (a canine and two bicuspid on each side) and fourteen lower teeth (one incisor, a canine, one bicuspid, and two molars on each side), the front teeth are conical, the molars dwarfed and none had been extracted.

Presented by Mr. W. A. MAGGS.

B. SIZE.—The teeth in the permanent series which are most frequently abnormally large are the central maxillary incisors and the second lower bicuspid, whilst diminution in size occurs more often in the lateral incisors and the third molars in the maxillæ.

Too large.

52. A large temporary molar with long roots.

53. Models showing large central and lateral incisors causing irregularity in both upper and lower jaws. The upper laterals were removed to make room for the other teeth.

Presented by Mr. H. P. TAYLOR.

54. A large left lower lateral incisor with flexuous root.

Presented by Mr. F. NEWLAND-PEDLEY.

55. A long upper right canine whose length has been partially increased by exostosis.

56. Four large lower canine teeth, one of which has a bifurcated root.

57. Two upper molars with very large crowns, in one, part of the crown has been destroyed by caries, in the other, the complexity may possibly be due to the fusion of a supernumerary tooth with a molar of ordinary size.

Presented by Mr. F. NEWLAND-PEDLEY.

58. A large upper molar with four roots.

Presented by Mr. E. ASHBY.

59. Four large upper molars.

60. Two lower molars, with long roots.

Too small.

61. A tiny supernumerary tooth.

62. Five stunted central incisor teeth.

Presented by Mr. F. V. MACKENZIE.

63. A pair of upper central incisors with short roots and crowns, and having stomatitic markings.

Presented by Mr. J. PAYNE.

64. Model of a lower jaw, showing a dwarfed central incisor.

Presented by Mr. J. M. C. JACOBS.

65. Three stunted upper bicuspid teeth.

66. Models showing small but perfectly shaped second upper bicuspids; in the lower jaw a diminutive left lateral is seen, while the bicuspids are normal.

Presented by Dr. J. W. PARE.

LIST
OF
GENTLEMEN EDUCATED AT GUY'S HOSPITAL
WHO HAVE PASSED THE
EXAMINATIONS OF THE SEVERAL UNIVERSITIES, COLLEGES,
&c., &c.,
IN THE YEAR 1899.

University of Oxford.

Examination for the Degree of Doctor of Medicine.

HARRY COOPER.

*Final Examination for the Degrees of Bachelor of Medicine and
Surgery.*

F. O. STOEHR.

University of Cambridge.

Degree of Doctor of Medicine.

A. P. BEDDARD.

Final Examination for the Medical and Surgical Degrees.

Part I.

A. H. Davies.
C. H. Glenn.

J. A. Glover.
T. E. Holmes.
L. Wilkin.

J. G. Taylor.
D. B. Watson.

Part II.

A. C. Fry.

H. A. Gaitskell.
D. P. Watson.

F. Shufflebotham.

Second Examination for the Medical and Surgical Degrees.

Part II.

S. Child.

H. A. Cutler.

H. P. Wiltshire.

First Examination for the Medical and Surgical Degrees.

Part II.

B. H. Stewart.

Examination in Sanitary Science.

Parts I. and II.

S. Copley.		C. A. Lumley.		A. E. Porter.
J. H. Godson.		E. L. Parry-Edwards.		H. J. Spon.
		C. M. Vernon.		

University of London.

Examination for the Degree of Doctor of Medicine.

R. H. Ashwin.		A. J. Cleveland.		R. W. Mayston.
H. W. Bruce.		C. R. Hoigson.		H. J. Starling.

Examination for the Degree of Master in Surgery.

W. S. Handley.		W. T. Milton.
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Examination for the Degree of Bachelor of Surgery.

First Division.

P. Turner.

Obtained Honours.

Second Division.

A. H. Carter.		J. Howell.		W. G. Stewart.
C. T. Hilton.		G. N. Meachen.		

Examination for the Degree of Bachelor of Medicine.

May.

First Division.

A. H. Carter.

Second Division.

F. J. H. Cann.		A. E. Clarke.		H. E. C. Fox.
		P. McK. Wilmot.		

October.

First Division.

H. L. Eason.

C. T. Hilton.

Obtained Honours in Medicine and Forensic Medicine.

Second Division.

G. N. Meachen.		P. Turner.
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Obtained Honours in Forensic Medicine.

E. S. Hall.		H. M. Reeve.		W. G. Stewart.
A. G. Osborn.		J. Robertson.		

Intermediate Examination in Medicine.

January.

Entire Examination.

Second Division.

G. T. Collins.		H. B. Foster.		H. C. Keates.
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Excluding Physiology.

First Division.

M. A. Collins.

Second Division.

W. J. Davies.		A. C. H. Gray.		W. M. Robson.
H. A. Ehrlich.		C. B. Penny.		D. H. Trail.
G. Evans.		E. F. Reeve.		

Physiology only.

First Division.

S. J. Ormond.		A. Pearson.		A. C. Ransford.
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Second Division.

D. Forsyth.		O. Marriott.		M. J. Rees.
E. T. Jensen.		T. A. Matthews.		D. W. Smith.

July.

Honours Examination.

W. H. Bowen.

Obtained Honours in Anatomy, Physiology and Histology, Materia Medica and Pharmaceutical Chemistry.

M. Coplans.		A. E. H. Pakes.		L. E. Stamm.
-------------	--	-----------------	--	--------------

Obtained Honours in Physiology and Histology.

Entire Examination.

Second Division.

H. W. Brown.		N. N. A. Houghton.		F. L. Thomas.
M. W. Cohen.		W. G. Parker.		A. H. E. Wall.
J. T. Hicks.		C. H. Robertson.		

Excluding Physiology.

Second Division.

J. Evans.		D. S. Graves.		H. K. Lacey.
		F. C. Wetherell.		

Physiology only.

Second Division.

M. A. Collins.		W. J. Davies.		A. C. H. Gray
		W. M. Robson.		

Preliminary Scientific (M.B.) Examination.

January.

Entire Examination.

First Division.

D. Isaacs.

Biology.

P. A. S. Dyson.		P. A. Peall.		G. H. Rees.
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July.

Entire Examination.

Second Division.

R. P. Rowlands.		G. Russell.		G. W. Smith.
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Chemistry and Experimental Physics.

G. Hamilton.		A. M. Roome.
--------------	--	--------------

Biology.

A. E. F. Kynaston.		J. O. Musson.		C. H. Reinhold.
		R. O. Williams.		

Intermediate Examination in Science.

E. H. B. Milsom.

Intermediate Examination in Science and Preliminary Scientific Examination conjointly.

Second Division.

H. H. Carter.		R. Felton.		M. G. Louisson.
J. H. Clatworthy.		A. Leeming.		A. P. Piggott.

University of Durham.

Examination for the Degree of Doctor of Medicine.

C. H. Bryant.

Examination for the Degree of Doctor of Medicine for Practitioners of Fifteen Years' Standing.

R. H. Browne.		J. S. Hooker.		W. Makeig Jones.
		Alexander Lane.		

Final Examination for the Degrees of Bachelor of Medicine and Surgery.

R. Tilbury Brown. | H. W. Dudgeon.

Bachelor in Medicine.—Third Examination.

H. Braund.

Bachelor in Medicine.—Second Examination.

S. C. Clapham. | M. C. Wetherell.

Bachelor in Medicine—First Examination.

Chemistry and Physics and Elementary Anatomy and Biology.

C. M. Anthony. | B. Glendining. | M. C. Wetherell.
J. W. Caton. | C. B. Travers. |

Chemistry and Physics only.

S. C. Clapham. | H. C. Sturdy. | G. W. Smith.

Royal College of Physicians of London.

Admitted to the Membership.

F. E. Fremantle. | R. O. Moon. | E. Ivens Spriggs.
L. C. Panting. | T. F. Ricketts. | D. W. Samways.

Final Examination for the License.

January.

H. M. Berncastle.	A. Evans.	F. J. Nicholls.
F. W. Brook.	Evan Evans.	R. J. Pritchard.
A. H. Carter.	J. H. Jones.	F. L. Rae.
A. E. Clarke.	A. H. B. Kirkman.	E. W. S. Rowland.
V. J. Crawford.	T. H. W. Landon.	F. D. Turner.
P. J. Curtis.	J. Matthews.	H. S. Turner.
J. T. Dunston.	W. T. Milton.	F. E. Walker.

April.

W. L. Baker.	J. W. Ensor.	A. A. Miller.
E. H. Cragg.	J. Howells.	J. E. H. Parsons.
H. S. Crapper.	A. D. Lewis.	J. H. Roberts.
E. B. Dowsett.	J. T. M. McDougall.	H. C. Sturdy.
W. H. Edwards.	G. N. Meachen.	O. C. Worthington.

July.

G. Beley.	A. R. McLachlan.	A. R. Thomas.
V. T. C. Bent.	C. D. Outred.	O. B. Thomson.
W. R. Cazenove.	H. M. Reeve.	T. M. Walker.
H. R. H. Denny.	S. A. Ruzzak.	J. B. Walters.
E. A. Evans.	G. S. Simpson.	O. F. Watson.
W. B. Hope.	J. G. Taylor.	

October.

G. M. Brown.	R. Michell.	J. L. Payne.
T. W. S. Browne.	D. J. Munro.	A. W. Penrose.
W. W. Harrison.	J. F. Northcott.	E. R. Row.
P. C. P. Ingram.	A. H. Palmer.	W. G. Stewart.
H. Leader.	E. E. Parrett.	D. P. Watson.
	L. Wilkin.	

Royal College of Surgeons of England.

Final Examination for the Fellowship.

S. Copley.	J. Howell.	F. W. Robinson.
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First Examination for the Fellowship.

W. H. Bowen.	A. C. H. Gray.	F. H. Parker.
F. Curtis.	L. Gifford Nash.	O. W. Richards.
M. A. Collins.	N. P. Blake Odgers,	C. Tessier.
	F. L. Thomas.	

Final Examination for the Membership.

January.

H. M. Berncastle.	A. Evans.	F. J. Nicholls.
F. W. Brook.	Evan Evans.	R. J. Pritchard.
A. H. Carter.	J. H. Jones.	F. L. Rae.
A. E. Clarke.	A. H. B. Kirkman.	E. W. S. Rowland.
V. J. Crawford.	T. H. W. Landon.	F. D. Turner.
P. J. Curtis.	J. Matthews.	H. S. Turner.
J. T. Dunston.	W. T. Milton.	F. E. Walker.

April.

W. L. Baker.	J. W. Ensor.	A. A. Miller.
E. H. Cragg.	J. Howells.	J. E. H. Parsons.
H. S. Crapper.	A. D. Lewis.	J. H. Roberts.
E. B. Dowsett.	J. T. M. McDougall.	H. C. Sturdy.
W. H. Edwards.	G. N. Meachen.	O. C. Worthington.

July.

G. Beley.	A. R. McLachlan.	A. R. Thomas.
V. T. C. Bent.	O. D. Outred.	C. B. Thomson.
W. R. Cazenove.	H. M. Reeve.	T. M. Walker.
H. R. H. Denny.	S. A. Ruzzak.	J. B. Walters.
E. A. Evans.	G. S. Simpson.	O. F. Watson.
W. B. Hope.	J. G. Taylor.	

October.

G. M. Brown.	R. Michell.	J. L. Payne.
T. W. S. Browne.	D. J. Munro.	A. W. Penrose.
W. W. Harrison.	J. F. Northcott.	E. R. Row.
P. C. P. Ingram.	A. H. Palmer.	W. G. Stewart.
H. Leader.	E. E. Parrett.	D. P. Watson.
	L. Wilkin.	

Royal College of Surgeons of Edinburgh.

Examination for the Fellowship.

C. W. Booker.	R. B. Stamford.
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Society of Apothecaries of London.

Final Examination for the License.

G. H. Bedford.	H. M. Hardy.	R. G. W. Saint-Cedd.
L. D. B. Cogan.	H. C. Holden.	T. J. Vick.
A. G. C. Davies.	E. McD. Judge.	C. C. Worts.
F. Golding-Bird.	S. H. Longhurst.	H. B. Yorath.

MEDALLISTS AND PRIZEMEN.

JULY, 1900.

Open Scholarships in Arts.

Frank Thomas Herbert Wood, Roan School, Greenwich, £100.
Herbert Stanley Knight, Bancroft's School, Woodford Wells, £50.
Herbert Andrew Watney, Trent College, Derbyshire, Certificate.

Dental Students.

Osborne Black, Foyle College, Londonderry, £30.

Open Scholarships in Science.

Henry Francis Bell Walker, Stockport Grammar School, and private study, £150.
John Hunter Clatworthy, Guy's Hospital, £60.
Gerald Russell, Guy's Hospital, Certificate.
Harry Hunter Carter, Guy's Hospital, Certificate.

Scholarship for University Students.

Paul Norman Blake Odgers, Lincoln College, Oxford, £50.
Greer Edmond Malcomson, Owen's College, Manchester, Certificate.
Frederick Henry Parker, Pembroke College, Cambridge, Certificate.

Junior Proficiency Prizes.

Percy Reginald Bolus, £20.
Edwin Henry Britton Milsom, £12 10s. } equal.
Neville Ivens Spriggs, £12 10s. }
Kenneth Black, Certificate.
Reginald Larkin, Certificate.
Alexander Moxon Webber, Certificate.

The Michael Harris Prize for Anatomy.

Alexander Moxon Webber, £10.
Reginald Larkin, Certificate.
William Leigh Maule Day } equal Certificate.
Lionel Henry Moiser }

The Wooldridge Memorial Prize for Physiology.

Percy Reginald Bolus, £10
Neville Ivens Spriggs, Certificate.
Edwin Henry Britton Milsom, Certificate.

The Hilton Prize for Dissection (1899).

Harold Tipping, £2 10s. } equal.
Alfred Herbert Edwin Wall, £2 10s. }

*The Arthur Durham Prizes for Dissection.**First Year's Students.*

Edward Crosby Peers, £5.

Senior Students.

Alexander Moxon Webber, £15.

Geoffrey Carlisle, Certificate.

Frederick Rogerson, Certificate.

*Dental Prizes.**First Year's Students*

Gerald Hamilton Morris, £10.

Samuel William Isles, Certificate.

Second Year's Students (1899).

John Black, £15.

Francis Wilberforce Garman } equal Certificates.

Harold Maurice }

Practical Dentistry Prize.

Gerald Hamilton Morris, £5 } equal.

Arthur Henry Clogg, £5 }

Senior Proficiency Prizes.

Graham Scales Simpson, £20.

Edward Cohen, Certificate.

The Treasurer's Gold Medal for Clinical Surgery.

Graham Scales Simpson.

The Beaney Prize in Pathology (1899).

Caleb Thomas Hilton, £34.

Frank Shufflebotham, Certificate.

The Richard Bredin Prize for Clinical Study.

Caleb Thomas Hilton, £25.

THE PHYSICAL SOCIETY.

Honorary President.—SIR SAMUEL WILKS, Bart., M.D., LL.D., F.R.S.

Secretaries.—Mr. Bellingham Smith and Dr. Beddard.

Presidents.

A. H. Davies, B.A., C. T. Hilton, M.B., B.S., A. H. B. Kirkman, F. Shufflebotham, M.A., M.B., B.C., F. O. Stoehr, B.A., M.B., B.Ch., R. H. J. Swan, M.B., B.S., P. Turner, M.B., B.S., B.Sc., F. G. Gibson, M.A., F. G. Cross, H. S. French, B.A., F. E. Walker.

PRIZEMEN FOR THE SESSION 1899-1900.

The Society's First Prize of £10 was awarded to Mr. F. G. Gibson for his paper on "The Ætiology and Prognosis of Epithelioma of the Tongue," and Mr. F. Cross obtained the Second Prize of £5 for his paper on "Abdominal Injuries."

CLINICAL APPOINTMENTS HELD IN THE YEAR 1899.

HOUSE PHYSICIANS.

F. E. Fremantle.
C. Jephcott.
L. E. C. Handson.

J. M. F. Brickdale.
E. W. S. Rowland.
W. H. M. Telling.

R. T. Fitz-Hugh.
K. B. Alexander.

HOUSE SURGEONS.

H. W. Bruce.
L. H. McGavin.
R. H. J. Swan.

W. T. Milton.
G. C. Owsley.
W. L. Baker.

F. E. Walker.
F. D. Turner.

ASSISTANT HOUSE SURGEONS.

W. J. Lindsay.
W. K. Wills.
T. H. W. Landon.
D. J. Munro.
A. A. Miller.
J. G. Taylor.

K. B. Alexander.
T. P. Berry.
A. H. B. Kirkman.
F. W. Brook.
A. R. Thomas.

A. H. M. Saward.
H. L. Eason.
A. H. Carter.
G. N. Meachen.
J. T. Dunston.

RESIDENT OBSTETRIC ASSISTANTS.

P. W. Moore.
F. S. Batchelor.
R. Balderston.
H. W. Bruce.

S. E. Denyer.
P. Turner.
T. P. Berry.
A. H. M. Saward.

A. J. Cleveland.
H. Leader.
W. J. Lindsay.

CLINICAL ASSISTANTS.

R. Balderston.	W. H. M. Telling.	H. L. Eason.
R. H. J. Swan.	T. P. Berry.	L. E. C. Handson.
D. J. Munro.	F. D. Turner.	J. G. Taylor.
J. T. Dunston.	W. L. Baker.	A. A. Miller.
A. H. M. Seward.	A. E. Clarke.	G. S. Simpson.
F. O. Stoehr.	D. P. Watson.	H. A. Gaitskell.
W. B. Secretan.	L. Wilkin.	R. C. Mullins.
E. Cohen.	J. F. Northcott.	G. N. Meachen.

CLINICAL ASSISTANTS IN THE MEDICAL WARDS.

H. Durbridge.	F. O. Stoehr.	F. W. Brook.
A. E. Clarke.	J. H. Roberts.	H. A. Gaitskell.
R. C. Mullins.	A. Fraser.	G. M. Brown.
W. G. Stewart.	A. H. Davies.	F. Shuffiebotham.
W. H. Brailey.	T. J. Wright.	H. N. Clarke.
C. B. Thomson.		

CLINICAL ASSISTANTS IN THE SURGICAL WARDS.

A. E. Cawston.	T. J. Wright.	J. Matthews.
W. M. Thomas.	C. D. Bryan.	W. H. Brailey.
J. F. Robinson.	P. W. L. Camps.	A. C. Ransford.
A. Pearson.		

SURGEONS' DRESSERS.

A. H. Davies.	F. J. Felix Jones.	A. C. Lewis.
H. B. Carr.	E. H. Felton.	A. McC. Dallas.
H. V. Bagshawe.	H. A. Higgins.	E. A. Miller.
W. H. Brailey.	T. P. Thomas.	P. D. Hunter.
J. F. Northcott.	R. C. Mullins.	H. B. Foster.
J. Atkins.	J. C. J. Da Silva.	J. S. S. Perkins.
R. M. Barron.	E. Stott.	A. Fraser.
E. Cohen.	G. Lewin.	T. E. Holmes.
T. T. Kelly.	J. E. L. Bates.	W. P. Ker.
A. C. Lewis.	J. M. Brydone.	J. A. Glover.
E. P. Mitchell.	E. T. Jensen.	W. Johnson.
F. G. Cross.	F. A. Segreda.	J. L. Whatley.
D. G. Greenfield.	R. D. Attwood.	T. R. Beale Browne.
E. J. F. Hardenberg.	H. McD. Parrott.	R. Tilbury.
P. S. Mandy.	E. G. Andrews.	A. Moon.
T. B. Fawley.	L. Pern.	H. G. Rashleigh.
E. F. Reeve.	W. H. Loosely.	B. Muir.
J. A. Wood.	R. S. Roper.	G. T. Willan.
P. W. L. Camps.	A. C. Ransford.	J. A. Butler.
A. Pearson.	F. G. Gibson.	D. Forsyth.
D. L. Morgan.	J. F. Robinson.	T. A. Matthews.
T. H. B. Dobson.	F. E. Welchman.	G. Clarke.
H. Wachter.	J. D. Bridger.	E. Shelton Jones.
J. A. B. Hammond.	E. C. Beavers.	E. G. Wales.
K. V. Trubshaw.	S. Hodgson.	S. J. Ormond.
F. W. Smith.	N. F. Ticehurst.	P. T. Manson.
G. S. Graham Smith.	H. Bentley.	H. Davies-Colley.

ASSISTANT SURGEONS' DRESSERS.

D. G. Greenfield.	C. R. Howard.	R. Tilbury.
J. M. Brydone.	H. McD. Parrott.	J. A. B. Hammond.
T. T. Kelly.	E. J. F. Hardenberg.	E. Shelton Jones.
J. L. Whatley.	F. A. Beattie.	T. H. B. Dobson.
F. A. Segreda.	G. Clarke.	S. J. Ormond.
E. H. Cragg.	J. E. L. Bates.	J. A. Glover.
R. D. Attwood.	E. T. Jensen.	F. G. Cross.
F. Curtis.	T. R. Beale Browne.	E. C. Bevers.
A. Moon.	W. P. Ker.	E. F. Reeve.
F. D. Welch.	P. W. L. Camps.	W. H. Loosely.
L. Pern.	J. A. Wood.	G. T. Willan.
T. B. Fawley.	H. G. Rashleigh.	J. A. Butler.
P. S. Mandy.	B. Muir.	A. C. Ransford.
D. Forsyth.	R. S. Roper.	F. G. Gibson.
K. W. Goadby.	A. Pearson.	H. Davies-Colley.
P. T. Manson.	J. D. Bridger.	H. Bentley.
D. L. Morgan.	N. F. Ticehurst.	K. V. Trubshaw.
E. G. Wales.	G. S. Graham Smith.	R. P. Marshall.
D. W. Smith.	R. Jimenez.	H. K. Lacey.
J. A. Matthews.	S. L. Prall.	F. M. Ommanney.
M. D. Wood.	P. J. Nash.	F. W. Smith.
H. Wachter.	S. Hodgson.	F. E. Welchmann.
H. S. French.	F. W. Sime.	E. H. Kitchen.
F. B. Manser.	E. F. G. Heap.	D. R. T. Griffith.
J. E. Collins.	R. D. Smedley.	S. S. H. Shannon.
R. Thompson.	W. O. Roberts.	H. D. Kempthorne.
V. M. Wallis.	L. Hirsch.	H. A. Ehrlich.
L. E. Stamm.	E. I. Claxton.	G. G. Davidson.

DENTAL SURGEONS' DRESSERS.

R. Howard.	H. R. H. Denny.	S. A. Ruzzak.
F. W. Brook.	H. M. Hardy.	T. E. Holmes.
E. E. Parrett.	O. Marriott.	B. W. Moss.
C. H. Brangwin.	H. Braund.	

CLINICAL ASSISTANTS IN MEDICAL OUT-PATIENTS.

J. G. Taylor.	G. N. Meachen.	W. H. Edwards.
W. B. Hope.	E. E. Parrett.	P. C. P. Ingram.
T. J. Wright.	E. J. Tongue.	E. A. Miller.
J. M. Brydone.	H. B. Dismorr.	E. H. Felton.

DRESSERS IN THE EYE WARDS.

E. J. Tongue.	E. R. Row.	L. Wilkin.
H. N. Clark.	G. H. Bedford.	H. A. Gaitskell.
C. B. Thomson.	A. A. Ruzzak.	R. Michell.
C. H. Brangwin.	J. F. Northcott.	J. Matthews.
C. T. Hilton.	A. Reid.	P. C. P. Ingram.
R. M. Barron.	L. Hirsch.	H. A. Higgins.
E. T. Jensen.	P. H. Ward.	H. T. Palmer.
A. H. B. Kirkman.	R. Tilbury.	G. Lewin.
R. D. Attwood.	H. McD. Parrott.	J. L. Whatley.

CLERKS IN THE EYE WARDS.

C. B. Sells.	H. R. H. Denny.	E. E. Parrett.
E. B. Dowsett.	G. N. Meachen.	H. M. Reeve.
H. M. Hardy.	F. O. Stoehr.	A. H. Carter.
H. E. C. Fox.	H. St. A. Alder.	T. H. W. Landon.
P. C. P. Ingram.	W. B. Secretan.	R. W. B. Hall.
A. Densham.	M. J. Rees.	H. Bentley.
F. W. Brook.	L. C. Martin.	C. H. Glenn.
J. B. C. Brockwell.	G. T. Wrench.	L. Hirsch.
W. W. Harrison.	M. W. Cohen.	H. Braund.
C. H. Robertson.	J. C. Curtis.	A. H. E. Wall.
C. B. Penny.	H. T. Palmer.	W. C. Lewis.
E. G. Andrew.	B. W. Moss.	F. L. Thomas.
J. A. Wood.		

DRESSERS IN THE THROAT DEPARTMENT.

T. W. H. Landon.	F. S. Batchelor.	A. A. Miller.
G. E. Richmond.	A. H. B. Kirkman.	E. W. S. Rowland.
H. C. Sturdy.	F. W. Brook.	C. T. Hilton.
J. Matthews.	C. G. Osborn.	J. Harris.
R. B. Stamford.	A. D. Lewis.	E. A. Longhurst.
A. R. McLachlan.	E. Cohen.	J. G. Taylor.
C. R. Hodgson.	H. J. Starling.	O. Marriott.
E. E. Parrett.	H. R. H. Denny.	C. E. Hicks.

CLERKS IN THE THROAT DEPARTMENT.

H. C. Sturdy.	W. B. Hope.	H. Braund.
G. T. Willan.	F. D. Welch.	C. B. Thomson.
W. O. Roberts.	L. Pern.	

MEDICAL WARD CLERKS.

A. C. Ransford.	J. A. Butler.	E. G. Allport.
D. Forsyth.	F. G. Gibson.	P. W. L. Camps.
E. F. Reeve.	R. S. Roper.	A. Pearson.
J. A. Wood.	G. S. Graham Smith.	H. Bentley.
K. V. Trubshaw.	H. Davies-Colley.	J. F. Robinson.
D. L. Morgan.	N. F. Ticehurst.	P. T. Manson.
J. E. Collins.	S. Hodgson.	M. D. Wood.
T. A. Matthews.	E. G. Wales.	H. K. Lacey.
R. Jimenez.	H. D. Kempthorne.	L. Hirsch.
P. J. Nash.	F. M. M. Ommanney.	R. P. Marshall.
S. S. H. Shannon.	D. W. Smith.	A. C. Osburn.
H. Wachter.	F. E. Welchman.	F. W. Smith.
J. A. B. Hammond.	D. R. T. Griffiths.	F. W. Sime.
T. H. F. Roberts.	S. J. Ormond.	H. A. Ehrlich.
E. C. Bevers.	V. M. Wallis.	C. B. Penny.
G. G. Davidson.	G. Clarke.	W. O. Roberts.
C. R. Howard.	T. H. B. Dobson.	E. I. Claxton.
E. H. Kitchin.	R. Thompson.	H. S. French.
A. Wylie.	F. B. Manser.	E. F. G. Heap.
C. B. Penny.	S. J. Ormond.	R. D. Smedley.
G. T. Collins.	A. Croneen.	L. E. Stamm.
C. E. Gaitskell.	F. Curtis.	F. A. Beattie.
M. J. Rees.	F. D. S. Jackson.	G. Evans.
J. F. Douse.	D. H. Trail.	H. C. Keates.
G. S. C. Hayes.	J. Evans.	S. C. H. Bent.
A. W. Soper.	E. Roberts.	A. W. Gater.
E. J. Crew.	A. C. Nash.	W. M. Robson.
G. H. H. Manfield.	H. J. Gater.	L. J. Hughes.
H. W. Brown.	F. C. Wetherell.	G. T. Wrench.
A. C. H. Gray.	M. A. Collins.	W. J. Davies.
C. Tessier.	G. W. C. Hollist.	H. A. Cutler.
R. C. Lawry.	E. Bigg.	J. A. Andrews.
S. Child.	H. P. Wiltshire.	O. W. Richards.

EXTERN OBSTETRIC ATTENDANTS.

J. Atkins.	W. H. Brailey.	E. A. Miller.
E. Cohen.	W. M. Thomas.	G. Shorland.
L. C. Martin.	C. T. Hilton.	G. S. Simpson.
W. G. Stewart.	R. W. B. Hall.	D. P. Watson.
W. A. Alabone.	E. W. Goble.	W. B. Secretan.
C. H. Glenn.	B. W. Moss.	E. G. Andrew.
T. Morgan.	C. A. D. Bryan.	A. Densham.
E. Shelton Jones.	A. W. Penrose.	T. J. Wright.
H. B. Carr.	A. E. Cawston.	A. H. Davies.
H. C. Holden.	B. Instone.	H. A. Higgins.
A. W. Talbot.	H. Braund.	E. H. Felton.
E. Stott.	H. B. Foster.	J. F. Northcott.
T. E. Holmes.	J. S. S. Perkins.	T. P. Thomas.
R. C. Mullins.	J. A. Glover.	G. Lewin.
A. C. Lewis.	E. P. Mitchell.	R. D. Attwood.
F. D. Welch.	K. W. Goadby.	T. T. Kelly.
W. P. Ker.	J. L. Whatley.	F. A. Segreda.
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E. T. Jensen.	W. H. Loosely.	J. M. Brydone.
J. E. L. Bates.	G. T. Willan.	

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W. M. Thomas.	B. Muir.	

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J. E. Collins.	H. Braund.	W. H. Brailey.
T. E. Holmes.		

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R. P. Marshall.	F. W. Sime.	A. C. Osburn.

CLERKS IN THE ELECTRICAL DEPARTMENT.

H. Braund.	D. L. Morgan.
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J. Bennett.	H. N. Hillier.	

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E. W. Corfe.	T. Walkington.	

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W. E. Lowe.	G. H. Morris.	S. W. Iles.
O. H. Dignum.	E. J. Gaffney.	S. C. Bowle.
H. B. Ross.	V. E. Turner.	T. Robinson.
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Consulting Obstetric Physician.—H. OLDHAM, M.D.

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<i>Morbid Histology and Bacteriology</i>	...	MR. BELLINGHAM SMITH, MR. PAKES.
<i>Medical and Surgical Pathology</i>		
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<i>Materia Medica and Therapeutics</i>	...	DR. PERRY.
<i>Practical Pharmacy</i>	THE HOSPITAL DISPENSER.
<i>Chemistry</i>	MR. WADE.
<i>Practical Chemistry</i>	MR. WADE, MR. RYFFEL AND MR. BALL.
<i>Experimental Physics</i>	PROFESSOR REINOLD, F.R.S., AND MR. BALL.
<i>Biology</i>	DR. STEVENS AND MR. SWAN.
<i>Psychology</i>	DR. SAVAGE AND DR. HYSLOP.

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For Prospectus and further information, apply to the Dean, Dr. FAWCETT, Guy's Hospital, London Bridge, S.E.

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C. S. MORRIS, L.D.S.E.

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L.D.S.E.

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Dental Anatomy and Physiology.—Mr. MAGGS.

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Curator of Dental Museum.—Mr. PAYNE.

Dean.—Dr. FAWCETT.

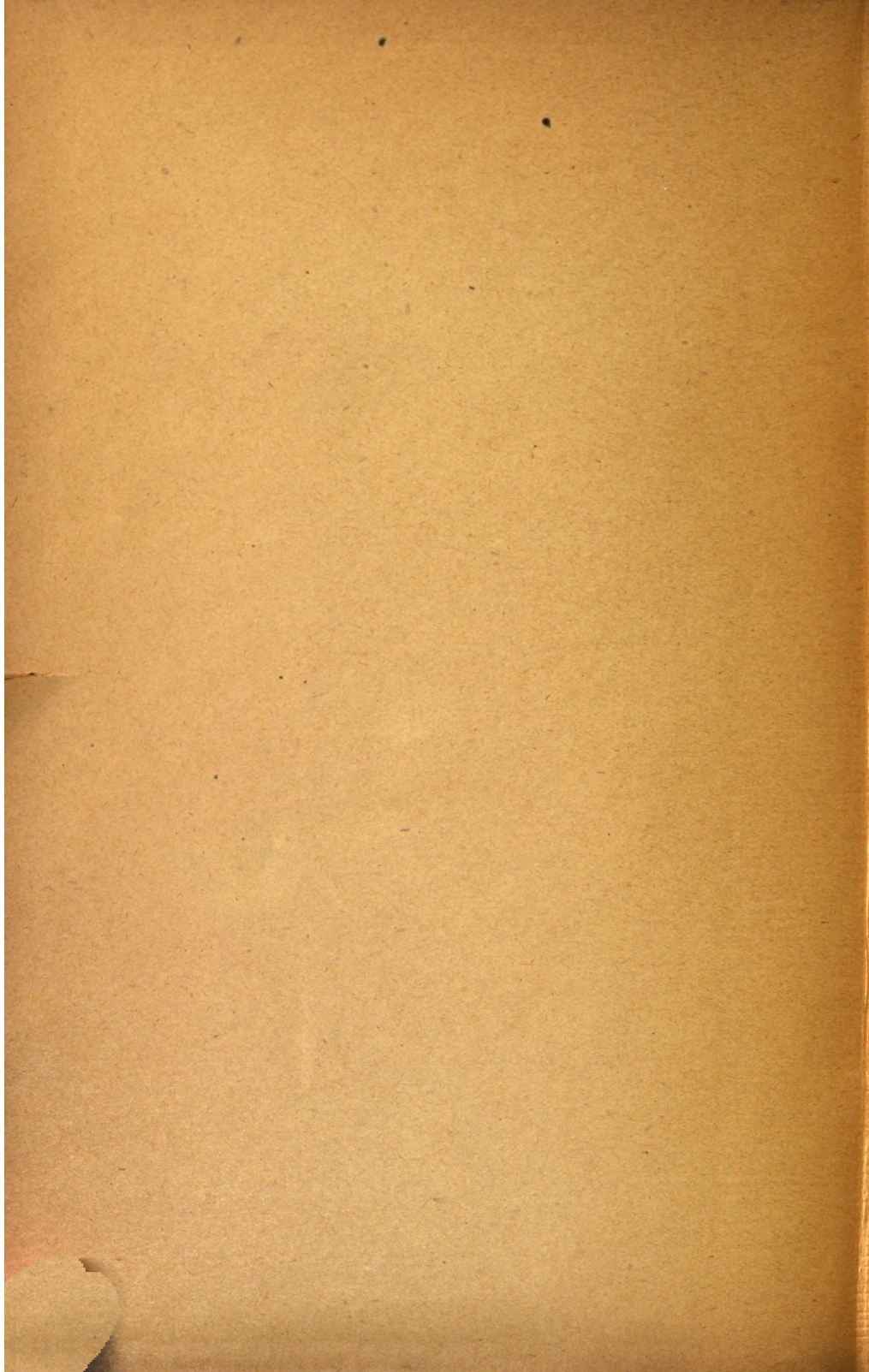
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J & A. CHURCHILL, Great Marlborough Street.



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